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United States Department of the Interior

FISH AND WILDLIFE SERVICE
300 WESTGATE CENTER DRIVE
HADLEY, MA 01035-9589

In Reply Refer To:
FWS/Region 5/ES-EC

MAY 15 1998

Memorandum

To: Chief, Division of Environmental Contaminants

From: Assistant Regional Director - Ecological Services

Subject: Transmission of Historic Completion Report - Metal and Organics Survey of Fish from the Connecticut River in New Hampshire (Joint Report PHS/FWS/89-2)

Attached is the final report by the New England Field Office for the subject on-refuge investigation copied from Region 5 archives. Funding for the study came from off-refuge Investigation 89-5-054. This study is shown in the National database having a report "DUE"; please change the entry to "COMPLETE".

As always, we welcome the opportunity to document the effectiveness and ingenuity of the Region 5 Field Contaminants Specialists. This report is an indicator of the level of imagination, effort, and perseverance invested in these studies, and reveals the extensive contributions made by the Environmental Contaminants Specialists in providing assistance to other Service programs, preventing injury to our resources, and giving early warning of impending problems in Region 5.

If you have any further questions or need additional information, please call Tim Fannin at (413) 253-8646.

Attachment

Disclaimer

The chemical analyses data presented here are for samples belonging to the State of New Hampshire, and were determined in accordance with contractual agreements between the State of New Hampshire and Resource Analysts Incorporated. The quality assurance/quality control procedures conducted under the agreement between the State of New Hampshire and Resource Analysts Incorporated are consistent with the U.S. Environmental Protection Agency's Contract Laboratory Program, although they may not necessarily conform to the quality assurance/quality control requirements of the U.S. Fish and Wildlife Service.

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Executive Summary

The Connecticut River is considered to be an important economic, environmental, and recreational resource for four of the six New England States, including New Hampshire. Characterization of the Connecticut River is of special interest not only because of the assets the River has to offer, but also because there is increasing development of the basin and an accompanying potential for the release of contaminants. Therefore, as an initial screening effort, the New Hampshire Department of Public Health Services (DPHS) and the U.S. Fish and Wildlife Service (FWS) jointly undertook a survey to characterize contaminant levels in selected species of fish sampled at five different locations along the New Hampshire reach of the Connecticut River.

Smallmouth bass, yellow perch and white perch were collected from five, four and three sampling locations, respectively, in June and July of 1986. Walleye, white suckers and chain pickerel were each collected from one sampling location during the same period. For each sampling location, fillets (skin off) and carcasses were composited separately and according to species, and analyzed for heavy metals (cadmium, chromium, lead and mercury) and organic compounds (DDT and metabolites, polychlorinated biphenyls - PCBs, and polynuclear aromatic hydrocarbons - PAHs). Levels of contaminants were assessed for potential impacts on the various fish sampled, as well as for the estimated potential health risks they may pose to humans from consumption.

Generally, the level of each contaminant analyzed for in these fish were unremarkable and within ranges that have been observed in fish taken from other rivers within New England and other northeastern states. However, the concentrations of PCBs and cadmium at specific sampling locations were found to exceed acceptable levels for wildlife. The acceptable concentration for cadmium (0.1 ppm to protect against adverse reproductive effects) was exceeded

in smallmouth bass and white perch collected at West Lebanon. Also, the acceptable level for PCBs in fish, established by the National Academy of Science (0.5 ppm to protect fish-eating wildlife), was exceeded in several species sampled at Claremont and below the Ashuelot River, and in just one species (smallmouth bass) sampled at Brattleboro.

Contaminant levels in these Connecticut River fish were also assessed for their potential health risks to human populations. A quantitative risk assessment was conducted to estimate the potential health risk from carcinogenic and noncarcinogenic fish contaminants. Carcinogenic risks were estimated for PCBs, PAHs and DDT metabolites based on U.S. Environmental Protection Agency (EPA) cancer potency factors. Noncarcinogenic health risks were evaluated for the remaining chemicals by comparing estimated doses resulting from fish consumption to reference dose values (RfDs). Such health risks were estimated for two populations of fish consumers, including one group consisting of individuals who consume an "average" amount of fish (7.8 g/person/day), and another consisting of individuals who consume fish at a rate equivalent to that of an avid sports fisherman (48 g/person/day). Estimated exposure levels resulting from consumption by either the average consumer or by the avid sports fisherman were not found to pose any significant noncarcinogenic health risks.

Of the various carcinogenic chemicals that were analyzed, only PCBs were estimated to pose a potentially significant cancer risk to both the average consumer and the avid sports fishermen populations. Based on median measured PCB concentrations, the estimated cancer risks to average and avid sports fishermen consumers are approximately 2 out of 10,000 and 2 out of 1,000, respectively. While the other two groups of carcinogenic contaminants (DDT/metabolites and PAHs) could possibly pose a carcinogenic health risk, the

data do not support that either were present at excessive levels, and the cancer risks that were calculated for these contaminants are likely overestimates due to incorporation of conservative assumptions (e.g., estimated contaminant concentrations when reported as less than detection were assumed to be equal to the detection limit).

All PCB concentrations detected in fish composites were below the FDA tolerance level of 2 ppm, and none of the PCB levels were observed to be any higher than those reported for fish sampled from other rivers in northeastern states. Therefore, the DPHS does not believe that the results from this study warrant a fish consumption advisory at this time. However, we do recommend that certain precautionary measures be taken in preparing these fish to help reduce the potential risks from exposure to contaminants such as PCBs. The DPHS recommends when preparing the fish to trim away areas with the highest potential PCB content, including the skin, fat belly meat, and dark fat along the backbone and lateral line.

For those who do consume fish from the Connecticut River, Table XVI provides the estimated increased risk of cancer from PCB exposure associated with various frequencies of meal consumption. The risks provided in Table XVI would be reduced if consumption occurs for less than a lifetime or if the fish preparation precautions, presented above, are followed.

The most remarkable fillet PCB composite concentration (1.64 ppm) was observed in walleye collected near Hanover. Since this was the only location where walleye were sampled, this may not be a representative sample for walleye living in other areas of the Connecticut River. Therefore, if resources become available, we recommend that further walleye sampling be conducted at a

representative number of locations to verify whether this single composite sample is representative of walleye in other areas of the River. Future sampling for the other species, as well, at all five locations would be desirable in order to characterize whether any trends in contaminant levels may be occurring.

SECTION 1 - WILDLIFE RISK ASSESSMENT

1.1 - INTRODUCTION

The Connecticut River originates in the Third Connecticut Lake in northern New Hampshire, and flows a total of 448 kilometers (280 miles) through New Hampshire, Vermont, Massachusetts and Connecticut. The basin's total area is 29,289 square kilometers (11,265 square miles) of which 7,953 square kilometers (3,059 square miles) are in New Hampshire.

Thirty entities in New Hampshire and 34 in Vermont are permitted to discharge effluents in the Connecticut river, while other contaminant loadings enter the river as non-point sources including hazardous waste sites, agricultural, and urban runoff.

In a continuing effort to characterize the waters in the State of New Hampshire and identify hazards to public health and wildlife, the New Hampshire Department of Public Health Services and the U.S. Fish and Wildlife Service jointly undertook a contaminant survey of fish in the New Hampshire reach of the Connecticut River.

Characterization of the Connecticut River is of special interest because of its importance as an economic, environmental, and recreational resource for four of six New England States, including New Hampshire, and because of increasing development of the basin, with its attendant potential for releases of contaminants. The survey described here was intended as a screening tool to identify problem areas and it is not intended to be comprehensive. More intensive surveys may be initiated to address site-specific contamination.

1.2 - METHODS AND MATERIALS

Fish were collected at five locations along the New Hampshire-Vermont portion of the river during the months of June and July of 1986. A second sample set was collected at one station in June of 1987 to complement the original sampling scheme. Figure 1 shows the location of the collection stations. The collecting sites were chosen to include areas downstream of major municipal and industrial development in the watershed, and as such, the data likely approximate worst case contaminant levels.

Variable mesh (50mm-75mm stretch) gill nets were used to collect fish. Fish were removed from nets within two hours of setting and were reset when necessary. Fish were selected according to availability, size, and likelihood of being kept by anglers. Weights and standard length of fish were recorded in the field prior to filleting.

Fillets were prepared as "skin off", wrapped in aluminum foil, put in polyethylene bags as composites according to species, and placed on ice. Carcasses were composited likewise.

Species retained for contaminant analysis were smallmouth bass (Micropterus dolomieui), yellow perch (Perca flavescens), white perch (Morone americana), walleye (Stizostedion vitreum), chain pickerel (Esox niger), and white sucker (Catostomus commersoni). Other species collected but released were rock bass (Ambloplites rupestris), pumpkinseed (Lepomis gibbosus), northern pike (Esox lucius), largemouth bass (Micropterus salmoides), brown bullhead (Ictalurus nebulosus), and golden shiner (Notemigonus crysoleucas).

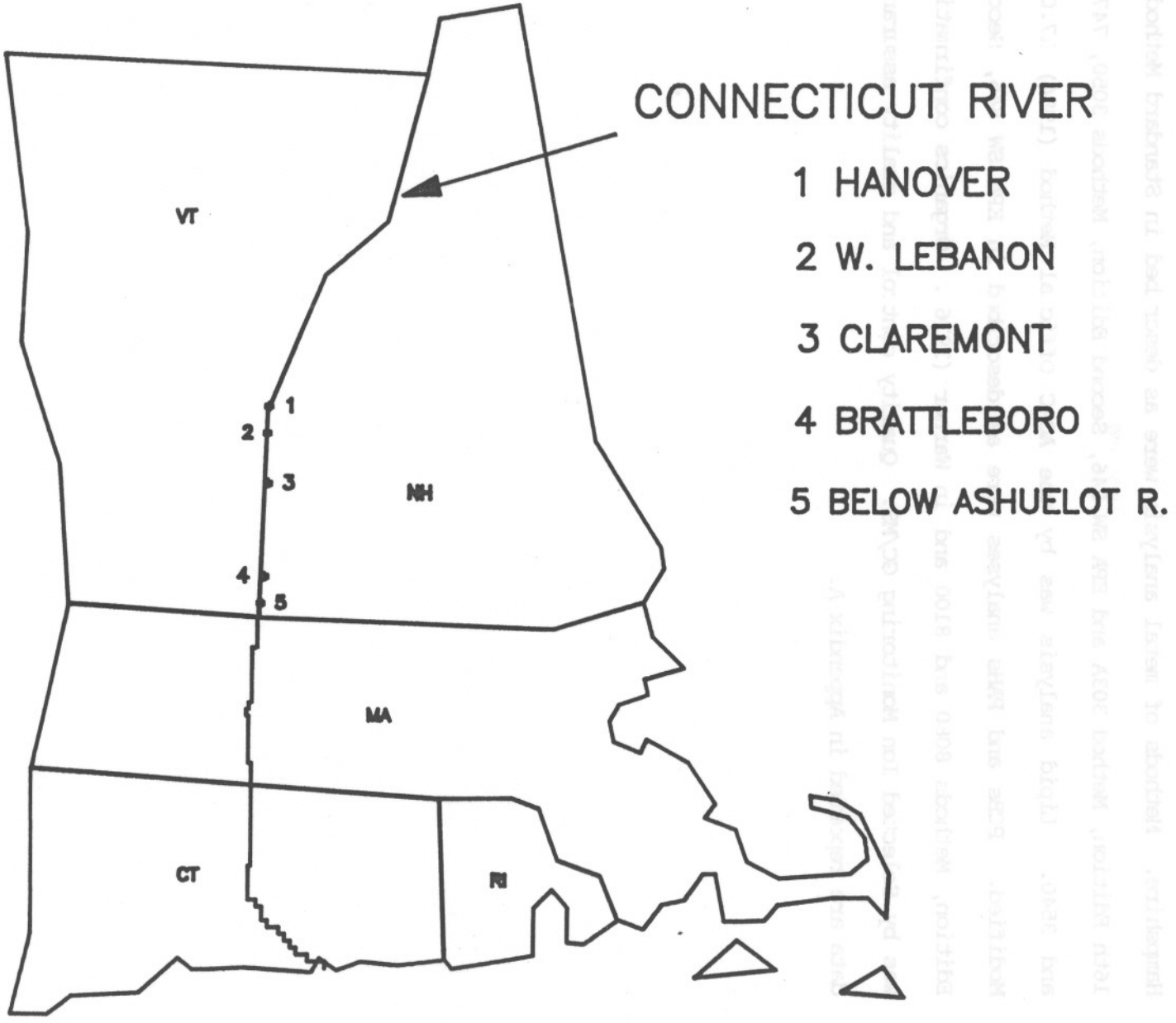

FIGURE 1. Locations of fish collection stations.

The samples were analyzed by Resource Analysts Incorporated of Hampton, New Hampshire. Methods of metal analysis were as described in Standard Methods, 16th Edition, Method 303A and EPA SW 846, Second Edition, Methods 3050, 7471, and 3540. Lipid analysis was by the AOAC Official Method (1984) 17.016 Modified. PCBs and PAHs analyses were as described in EPA SW 846, Second Edition, Methods 8080 and 8100 and in Warner (1976). Organics confirmation was by Selected Ion Monitoring GC/MS. Quality control and quality assurance data are reported in Appendix A.

1.3 - RESULTS

The results of chemical analyses are presented in Table-I and IA. Average values for the three species for which analyses were conducted appear in Table-II. Table-III represents weighted concentration values (Appendix B). These values take into consideration the total weight (fillet and offal) of tissue analyzed and their concentrations. All values are expressed in wet weight. Polynuclear Aromatic Hydrocarbon values are expressed as total PAHs and consist of one or more of the following compounds: phenanthrene, anthracene, fluoranthene, pyrene, and chrysene.

Generally, the levels of contaminants found are unremarkable. PCBs are the only contaminants surveyed that show elevated levels at some locations. Fillets of walleyes taken at Hanover and whole white perch taken near the confluence of the Ashuelot and Connecticut Rivers had PCBs in excess of 1.0 ppm, but less than 2.0 ppm.

Mercury and lead levels were below 0.33 ppm and 0.54 ppm, respectively, at all locations. PAH levels are less than 0.1 ppm except for a level of 0.23 ppm for whole yellow perch taken at West Lebanon. Cadmium and chromium levels were less than 0.18 ppm and 1.5 ppm, respectively.

TABLE 1 - CONCENTRATIONS OF METAL AND ORGANIC CONTAMINANTS IN CONNECTICUT RIVER FISH (UG/G WW)

STATION	ID NUMBER	SPECIES *	Cd	Cr	Pb	Hg	PCBs	PAHs	DDT	DDD	DDE
HANOVER	060486-1	Smallmouth Bass-F	0.034	0.600	0.130	0.290	0.110	---	<.02	<.02	0.020
	060486-4	Smallmouth Bass-W	0.011	0.980	0.540	0.150	0.380	---	<.02	<.02	0.080
	060486-2	Yellow Perch-F	0.046	1.300	0.084	0.270	0.140	---	<.02	<.02	0.020
	060486-5	Yellow Perch-W	0.020	1.100	0.270	0.160	0.330	---	<.02	<.02	0.070
	060486-3	Walleye-F	0.065	1.200	0.230	0.330	1.640	---	<.02	<.02	0.260
	060486-6	Walleye-W	0.012	0.650	0.150	0.180	0.210	---	<.02	<.02	0.030
W. LEBANON	072386-3	Smallmouth Bass-F	0.160	1.200	<.500	0.080	0.110	<.10	<.001	<.001	0.006
	072386-4	Smallmouth Bass-W	0.140	0.340	<.500	<.060	0.300	<.10	<.001	<.001	0.021
	061087-1	Yellow Perch-F	<.050	<.100	<.500	0.210	0.041	<.10	---	---	---
	061087-2	Yellow Perch-F	<.050	<.100	<.500	<.050	0.065	<.10	---	---	---
	061087-3	Yellow Perch-W	<.050	0.460	<.500	<.050	0.180	0.23	---	---	---
	072386-1	Yellow Perch-F	0.120	0.960	<.500	0.200	0.170	<.10	<.001	<.001	0.020
	072386-2	Yellow Perch-W	0.160	0.690	<.500	0.100	0.055	<.10	.012	<.001	<.001
	072386-5	White Perch-F	0.140	1.200	<.500	0.180	0.340	<.10	<.001	<.001	0.027
	072386-6	White Perch-W	0.180	0.750	<.500	0.130	0.140	<.10	<.001	<.001	0.051
CLAREMONT	060986-1	Smallmouth Bass-F	0.010	0.830	0.046	0.060	0.110	<.10	<.02	<.02	0.020
	060986-2	Smallmouth Bass-W	0.029	1.100	0.140	<.060	0.260	<.10	<.02	<.02	0.040
	060986-3	Yellow Perch-F	0.024	0.880	0.089	0.120	0.370	<.10	<.02	<.02	0.060
	060986-4	Yellow Perch-W	0.059	0.700	0.120	0.110	0.710	<.10	<.02	<.02	0.140
	060986-5	White Sucker-F	0.006	0.880	0.039	<.060	0.073	<.10	<.02	<.02	<.01
	060986-6	White Sucker-W	0.091	1.000	0.150	<.060	0.150	<.10	<.02	<.02	0.020
BRATTLEBORO	060386-1	Smallmouth Bass-F	0.018	0.079	0.092	0.130	0.670	<.10	<.02	<.02	0.080
	060386-2	Smallmouth Bass-W	0.018	0.990	0.170	0.110	0.580	<.10	<.02	<.02	0.080
	060386-3	Yellow Perch-F	0.050	1.300	0.120	0.130	0.160	<.10	<.02	<.02	0.020
	060386-4	Yellow Perch-W	0.014	1.500	0.170	0.080	0.410	<.10	<.02	<.02	0.070
	060386-5	White Perch-F	0.026	1.300	0.130	0.100	0.140	<.10	<.02	<.02	0.020
	060386-6	White Perch-W	0.026	1.300	0.160	0.090	0.540	<.10	<.02	<.02	0.080
BELOW ASHUELOT RIVER	060286-1	Chain Pickerel-F	0.022	1.100	0.160	0.230	0.031	<.10	<.02	<.02	<.04
	060286-2	Chain Pickerel-W	0.040	1.300	0.150	0.200	0.950	<.10	<.02	<.02	0.150
	060286-3	Smallmouth Bass-F	0.025	0.870	0.120	0.100	0.140	<.10	<.02	<.02	0.020
	060286-4	Smallmouth Bass-W	0.040	1.200	0.140	<.060	0.800	<.10	<.02	<.02	0.100
	060286-5	White Perch-F	0.025	0.880	0.120	0.190	0.690	<.10	<.02	<.02	0.140
	060286-6	White Perch-W	0.036	1.500	0.160	0.100	1.550	<.10	<.02	<.02	0.210

* F: fish fillet
skin off
W: remaining
carcass

TABLE I - A CONCENTRATIONS OF METAL AND ORGANIC CONTAMINANTS IN INDIVIDUAL SPECIES OF CONNECTICUT RIVER FISH (UG/G WW)

SPECIES *	Cd	Cr	Pb	Hg	PCBs	PAHs	DDT	DDD
Chain Pickerel-F	0.022	1.100	0.160	0.230	0.031	<.10	<.02	<.02
Chain Pickerel-W	0.040	1.300	0.150	0.200	0.950	<.10	<.02	<.02
Smallmouth Bass-F	0.160	1.200	<.500	0.080	0.110	<.10	<.001	<.001
Smallmouth Bass-F	0.034	0.600	0.130	0.290	0.110	---	<.02	<.02
Smallmouth Bass-F	0.018	0.079	0.092	0.130	0.670	<.10	<.02	<.02
Smallmouth Bass-F	0.025	0.870	0.120	0.100	0.140	<.10	<.02	<.02
Smallmouth Bass-F	0.010	0.830	0.046	0.060	0.110	<.10	<.02	<.02
Smallmouth Bass-W	0.029	1.100	0.140	<.060	0.260	<.10	<.02	<.02
Smallmouth Bass-W	0.011	0.980	0.540	0.150	0.380	---	<.02	<.02
Smallmouth Bass-W	0.040	1.200	0.140	<.060	0.800	<.10	<.02	<.02
Smallmouth Bass-W	0.018	0.990	0.170	0.110	0.580	<.10	<.02	<.02
Smallmouth Bass-W	0.140	0.340	<.500	<.060	0.300	<.10	<.001	<.001
Walleye-F	0.065	1.200	0.230	0.330	1.640	---	<.02	<.02
Walleye-W	0.012	0.650	0.150	0.180	0.210	---	<.02	<.02
White Perch-F	0.025	0.880	0.120	0.190	0.690	<.10	<.02	<.02
White Perch-F	0.140	1.200	<.500	0.180	0.340	<.10	<.001	<.001
White Perch-F	0.026	1.300	0.130	0.100	0.140	<.10	<.02	<.02
White Perch-W	0.180	0.750	<.500	0.130	0.140	<.10	<.001	<.001
White Perch-W	0.026	1.300	0.160	0.090	0.540	<.10	<.02	<.02
White Perch-W	0.036	1.500	0.160	0.100	1.550	<.10	<.02	<.02
White Sucker-F	0.006	0.880	0.039	<.060	0.073	<.10	<.02	<.02
White Sucker-W	0.091	1.000	0.150	<.060	0.150	<.10	<.02	<.02
Yellow Perch-F	0.050	1.300	0.120	0.130	0.160	<.10	<.02	<.02
Yellow Perch-F	0.120	0.960	<.500	0.200	0.170	<.10	<.001	<.001
Yellow Perch-F	<.050	<.100	<.500	0.210	0.041	<.10	---	---
Yellow Perch-F	0.046	1.300	0.084	0.270	0.140	---	<.02	<.02
Yellow Perch-F	0.024	0.880	0.089	0.120	0.370	<.10	<.02	<.02
Yellow Perch-F	<.050	<.100	<.500	<.050	0.065	<.10	---	---
Yellow Perch-W	0.014	1.500	0.170	0.080	0.410	<.10	<.02	<.02
Yellow Perch-W	0.020	1.100	0.270	0.160	0.330	---	<.02	<.02
Yellow Perch-W	0.160	0.690	<.500	0.100	0.055	<.10	.012	<.001
Yellow Perch-W	<.050	0.460	<.500	<.050	0.180	0.23	---	---
Yellow Perch-W	0.059	0.700	0.120	0.110	0.710	<.10	<.02	<.02

* F: fish fillet skin off
W: remaining carcass

TABLE II - AVERAGE CONCENTRATIONS OF CONTAMINANTS IN CONNECTICUT RIVER FISH (UG/G WW)

SPECIES *	Cd	Cr	Pb	Hg	PCBs	PAHs	DDT	DDD	DDE
SMALLMOUTH BASS-F	0.049	0.716	0.078	0.132	0.228	---	<.02	<.02	0.029
-W	0.480	0.922	0.198	0.052	0.464	<.100	<.02	<.02	0.064
WHITE PERCH-F	0.640	1.127	0.083	0.157	0.390	<.100	<.02	<.02	0.062
-W	0.081	1.183	0.107	0.107	0.743	<.100	<.02	<.02	0.114
YELLOW PERCH-F	0.040	0.740	0.490	0.155	0.158	0.150	<.02	<.02	0.020
-W	0.510	0.890	0.112	0.900	0.337	<.100	0.007	<.02	0.056

* F: fish fillet skin off
  W: remaining carcass

TABLE III - WHOLE BODY (WEIGHTED) METAL AND ORGANIC CONCENTRATIONS IN CONNECTICUT RIVER FISH (UG/G WW).

STATION	SPECIES	Cd	Cr	Pb	Hg	PCBs	DDT (Total)
Hanover	Smallmouth bass	0.014	0.932	0.488	0.168	0.346	0.072
W. Lebanon		0.143	0.451	0.5	0.077	0.276	0.019
Claremont		0.026	1.063	0.127	0.06	0.239	0.037
Brattleboro		0.018	0.855	0.158	0.113	0.593	0.080
Below Ashuelot Rv		0.038	1.152	0.151	0.204	0.704	0.088
Hanover	Yellow perch	0.023	1.126	0.246	0.174	0.306	0.064
W. Lebanon		0.036	0.199	0.5	0.086	0.114	*
Claremont		0.054	0.727	0.115	0.112	0.658	0.128
Brattleboro		0.02	1.469	0.162	0.088	0.371	0.062
W. Lebanon	White perch	0.175	0.807	0.5	0.136	0.166	0.048
Brattleboro		0.026	1.3	0.156	0.091	0.488	0.083
Below Ashuelot Rv		0.035	1.445	0.138	0.096	1.876	0.204
Hanover	Walleye	0.016	0.697	0.157	0.193	0.333	0.5
Claremont	White sucker	0.018	0.137	0.06	0.131	0.98	0.077
Below Ashuelot Rv	Pickerel	0.037	1.267	0.153	0.159	0.779	0.132

* Sample not tested

1.4 - DISCUSSION OF CONTAMINANT SOURCES, ENVIRONMENTAL FATE AND ANIMAL TOXICITY

1.4.1 - CADMIUM

Cadmium is a heavy metal naturally present in the environment in trace amounts. It has been estimated that 290 metric tons/yr are emitted from natural sources worldwide and that 5,500 metric tons/yr are emitted from anthropogenic sources (U.S. EPA, 1981). Cadmium, zinc, and lead smelting operations produce trace amounts of these elements which travel through the atmosphere, potentially affecting areas immediate to and remote from the source. Electroplating industries, phosphate fertilizers, and sulfide ore mining activities are associated with cadmium contamination (May and McKinney, 1981). Cadmium can enter aquatic environments through atmospheric deposition, point source and non-point source effluents.

According to Callahan et al (1979), photolysis, volatilization, and biotransformation do not appear to play an important role in the fate of cadmium. In polluted waters, it complexes with organic matter and sorption to suspended and bed sediments reduces its mobility. Biota strongly accumulate cadmium and more so in soft than in hard water.

Cadmium residues in freshwater biota are lower than in marine biota because of lower cadmium levels in freshwater (Eisler, 1985a). Cadmium is extremely toxic and bioaccumulates in fish, where the liver and kidneys are main targets even at low concentrations (Rompala et al, 1984; McFarlane and Franzin, 1980; Badsha and Goldspink, 1982; Ney and Van Hassel, 1983). Exposure to cadmium has resulted in renal damage (Nordberg et al, 1979; Gill and Pant, 1983), testicular atrophy (Nordberg, 1971; Sangalang and O'Halloran, 1972), and liver damage in both mammals and fish (Roberts et al, 1979; Lowe-Linde and Niimi, 1984; Rajana et al, 1985).

Cadmium also affects growth and can cause mortality in freshwater fish (Davis et al, 1977), may inhibit or restrict calcium metabolism, damage fish vertebral columns (Lockwood, 1976; Horne, 1978; Muramoto, 1981; Kanciruk, 1982), and cause fin erosion (Sinderman, 1979). Cadmium-induced abnormal behavior in fish suggests nervous system damage (Cearley and Coleman, 1974). Adverse reproductive effects may occur at 0.1 ppm tissue concentration (Sloan, 1983). This value was exceeded in smallmouth bass and white perch from West Lebanon, and yellow perch from Hanover.

TABLE IV - WEIGHTED CADMIUM CONCENTRATIONS IN FISHES

SPECIES	CONC. (UG/G) WW	LOCATION	REFERENCE
SMALLMOUTH BASS	ND - 0.050	PENNSYLVANIA	ROMPALA et al, 1984
	0.038	BELOW ASHUELOT RV.	PRESENT STUDY
	0.018	BRATTLEBORO	
	0.026	CLAREMONT	
	0.143	WEST LEBANON	
	0.014	HANOVER	
WHITE SUCKER	ND - 0.480	PENNSYLVANIA	ROMPALA et al,1984
	0.100	MERRIMACK RV.,MA	NCBP, 1984
	0.077	CLAREMONT	PRESENT STUDY
YELLOW PERCH	0.170	CONN. RV., CONN.	NCBP,1984
	0.020	ANDROSCOGGIN RV., ME	
	0.020	BRATTLEBORO	PRESENT STUDY
	0.054	CLAREMONT	
	0.036	WEST LEBANON	
	0.023	HANOVER	
WHITE PERCH	0.030	SUSQUEHANNA RV.,MD	NCBP,1984
	0.035	BELOW ASHUELOT RV.	PRESENT STUDY
	0.026	BRATTLEBORO	
	0.175	WEST LEBANON	
WALLEYE	0.016	HANOVER	PRESENT STUDY
CHAIN PICKEREL	0.037	BELOW ASHUELOT RV.	PRESENT STUDY

1.4.2 - CHROMIUM

Natural occurrence of chromium is as the ore, chromite. It is present in low concentrations throughout nature (EPA, 1986). According to Eisler (1986), most of it is produced by the USSR and South Africa, and the annual world production is estimated at 7 million metric tons. Most of the chromium in aquatic ecosystems comes from atmospheric emissions, metal industries, fertilizers, and sewage treatment plants (Ecological Analysts, 1981; Langar and Nortset, 1979; Towill et al, 1978).

According to Steven et al, (1976) chromium occurs in several oxidation states, with chromium VI and III being the most significant because they are more stable.

Photolysis, volatilization, biotransformation, and sorption are not very important processes in the environmental fate of chromium according to Callahan et al (1979). They found that speciation is important because it controls the intertransformation of chromium VI to chromium III.

As an essential nutrient (Steven et al, 1976), chromium is bioaccumulated by aquatic organisms. Chromium toxicity, especially when present in an aquatic environment, is affected by temperature, pH, salinity, water hardness, alkalinity, animal species, age, interaction with other toxicants, duration of exposure, and chromium form (Eisler, 1986a; Rompala et al, 1984). Non-lethal levels of chromium can express toxicity in other forms. For example, laboratory studies indicate that hexavalent chromium adversely affects the survival and growth of fish (Rompala et al, 1984). Adsorption and bioaccumulation are relatively minor processes in determining the fate of

chromium in aquatic environments (Ecological Analysts, 1981). Chromium accumulates mainly in the pyloric caeca, intestines, kidneys, and liver of fish (Rompala et al, 1984). The bioaccumulation of chromium and its impacts to food chains is relatively poorly understood. According to Eisler (1986a), there is little evidence of chromium magnification through food chains, although, Papadopoulu (1973) found biomagnification to occur in marine mollusks. Rompala et al (1984) state that some fish concentrate chromium 100 times that of ambient water levels. While the significance of chromium residues in biota is unclear, available evidence suggests that organs and tissues in fish and wildlife that contain greater than 4,000 ug Cr/kg D.W. (800 ug/kg W.W.) should be viewed as presumptive evidence of chromium contamination (Eisler, 1986a). Fish collected exceed this value at all stations in the Connecticut River except for smallmouth bass and yellow perch from West Lebanon.

TABLE V - WEIGHTED CHROMIUM CONCENTRATIONS IN FISHES

SPECIES	CONC. (UG/G) WW	LOCATION	REFERENCE
SMALLMOUTH BASS	1.152	BELOW ASHUELOT RV.	PRESENT STUDY
	0.855	BRATTTLEBORO	
	1.063	CLAREMONT	
	0.451	WEST LEBANON	
	0.932	HANOVER	
WHITE SUCKER	ND- 0.100	PENNSYLVANIA	ROMPALA et al, 1984
	0.980	CLAREMONT	PRESENT STUDY
YELLOW PERCH	1.469	BRATTLEBORO	PRESENT STUDY
	0.727	CLAREMONT	
	0.199	WEST LEBANON	
	1.126	HANOVER	
WHITE PERCH	1.455	BELOW ASHUELOT RV.	PRESENT STUDY
	1.300	BRATTLEBORO	
	0.807	WEST LEBANON	
WALLEYE	0.200	MISSOURI RV.,IA	STEVENS AND TONDREAU, 1986
	0.697	HANOVER	PRESENT STUDY
CHAIN PICKEREL	1.267	BELOW ASHUELOT RV.	PRESENT STUDY

1.4.3 - LEAD

Lead is a major constituent of more than 200 identified minerals, of which only three are abundant: galena, angelesite, and cerrusite (U.S. EPA, 1979). Lead production in 1980 was 5,100 metric tons, with its major uses including: storage batteries, gasoline, cable covering, solder, ammunition, metal industry, and chemicals (Demayo et al,1981; Moore and Ramamoorthy, 1983). The global lead input into aquatic environments has been estimated between 630,000 and 770,000 metric tons a year by Demayo et al (1981). While Callahan et al (1979) suggest gasoline combustion as one of the major sources of lead being released into the environment, May and McKinney (1977) list lead mining, smelting operations, industrial effluents, atmospheric fallout from coal burning stations, landfills, and sewage sludge as major lead sources in the environment.

Lead tends to complex with naturally occurring organic materials like humic and fulvic acids and can be methylated by benthic organisms according to Callahan et al (1979). They concluded that once methylated, lead is more volatile and toxic to organisms than elemental lead, which is insoluble in water but may become soluble under acidic conditions. In aquatic systems, lead is removed from solution and suspension, and sorbed to the sediments (Helz et al, 1975; Valiela et al, 1974). Geological conditions, pH, hardness, complexing agents, iron concentrations, and sediment type are mechanisms which control lead concentration in water and thus its availability to biota (Callahan et al, 1979).

Chemical speciation determines which solid species controls solubility in unpolluted waters and its organic complexation is an important mechanism for its fate in polluted waters. Lead sorption to organic and inorganic materials control its mobility. Biotransformation and bioaccumulation are important mechanisms in the environmental fate of lead.

Except for Moore and Ramamoorthy (1983), most authors agree that lead is not biomagnified and its bioconcentration factors decrease as trophic levels increase (Callahan et al, 1979; Demayo et al, EPA, 1980). Chronic toxicity studies have shown lead accumulates mostly in the kidneys and gills, and to a lesser extent in the livers of fish (Holcombe et al, 1976). Little lead is accumulated in finfish muscle, but bivalve shellfish can accumulate lead to high levels (Callahan et al, 1979; Merlin and Pozzi, 1977; Holcombe et al, 1976; Phillips and Russo,1976). Chronic toxicity studies of lead's effect in some freshwater fish species show an increase in spinal cord deformities (Holcombe et al, 1976). According to EPA (1980c), delayed embryonic development, suppressed reproduction, and inhibition of growth rate in fish are caused by lead, with adverse effects occurring at concentrations as low as 25 ppb. Moore and Ramamoorthy (1983) concluded that chronic effects of lead in invertebrates may or may not appear below the LC-50 value (concentration lethal to 50% of the organisms). Therefore, in aquatic systems, the presence of invertebrates near the source of lead does not give an accurate account of contamination because its chronic impacts could appear abruptly.

Phillips and Russo (1978) suggest that fish livers exceeding 50 ug Pb/g and kidneys above 180 ug Pb/g may be an indication of unacceptable exposure. It has been suggested that lead values in fish higher than 1 ppm may represent a contaminant problem (Rompala et al, 1984). This level was not observed in any of the Connecticut River fish samples.

TABLE VI - WEIGHTED LEAD CONCENTRATIONS IN FISHES

SPECIES	CONC. (UG/G) WW	LOCATION	REFERENCE
SMALLMOUTH BASS	ND- 0.510	PENNSYLVANIA	ROMPALA et al, 1984
	0.100	PENOBSCOT RV.,ME	NCBP, 1984
	0.151	BELOW ASHUELOT RV.	PRESENT STUDY
	0.158	BRATTLEBORO	
	0.127	CLAREMONT	
	<0.500	WEST LEBANON	
	0.488	HANOVER	
WHITE SUCKER	ND - 1.200	PENNSYLVANIA	ROMPALA et al, 1984
	0.110	PENOBSCOT RV.,ME	NCBP, 1984
	0.380	MERRIMACK RV., MA	NCBP, 1984
	0.060	CLAREMONT	PRESENT STUDY
YELLOW PERCH	0.400	CONNECTICUT RV.,CT	NCBP, 1984
	2.020	LAKE ERIE, PA	
	0.250	ANDROSCOGGIN RV.,ME	
	0.162	BRATTLEBORO	PRESENT STUDY
	0.115	CLAREMONT	
	<0.500	WEST LEBANON	
	0.246	HANOVER	
WHITE PERCH	0.450	SUSQUEHANNA RV.,MD	NCBP, 1984
	0.138	BELOW ASHUELOT RV.	PRESENT STUDY
	0.156	BRATTLEBORO	
	<0.500	WEST LEBANON	
WALLEYE	2.340	MISSORI RV.,IA	STEVENS AND TONDREAU, 1986
	0.157	HANOVER	PRESENT STUDY
CHAIN PICKEREL	0.153	BELOW ASHUELOT RV.	PRESENT STUDY

1.4.4 - MERCURY

The major natural source of mercury is the natural degassing of the earth's crust which is estimated to release between 20,000 and 120,000 metric tons of mercury a year worldwide (WHO, 1976). According to the 1976 WHO report, anthropogenic sources of mercury contribute about 20,000 metric tons per year to the environment, which is less of the mercury load than attributable to natural sources.

In natural waters, mercury can exist in several forms. The mercury species depends on pH, sediment redox potential, hardness, alkalinity, and concentrations of anions (Moore and Ramamoorthy, 1983; Akielaszek and Haines, 1981). Adsorption to particulate matter and sedimentation are mechanisms which remove mercury from solution in natural waters (Callahan et al, 1979). Inorganic mercury can be methylated by bacteria in sediments, entering the aquatic food chain and accumulating in biota (WHO, 1976), a condition enhanced by nutrient enrichment (Jernelov, 1972). According to Moore and Ramamoorthy (1983), mercury methylation rates in freshwater systems are higher than in marine systems. They also suggest that because freshwater systems are more confined than marine systems, mercury concentrations in freshwater biota are higher.

According to Callahan et al (1979), photolysis of mercury may be important in some aquatic environments. Chemical speciation controls its volatility by conversion of metallic mercury to complexed species. Volatilization is important in mercury movement in and out of aquatic environments. Sorption results in partitioning of mercury into suspended and bed sediments and it is strongest into organic material. Bioaccumulation occurs especially with the methylated forms of mercury. Biotransformation by bacteria to its methylated forms makes it quite mobile.

Factors believed to affect mercury toxicity include age, surface area of the organism, metabolism, habitat, and activity (Callahan et al, 1979). The most toxic mercury species is methylmercury (Callahan et al, 1979; Moore and Ramamoorthy, 1983; EPA, 1984; WHO, 1976; Eisler, 1987a) and its biological half life is between one and three years (Phillips and Russo, 1978).

Mercury and its compounds have no known biological function and are potential hazards to living organisms according to Eisler (1987a). He also suggests that low-toxicity inorganic mercury can become highly toxic through biological processes and can be bioconcentrated and biomagnified through the food chain. The kidney is the main target of mercury in animals (WHO, 1976) and is eliminated through urine and feces.

Moore and Ramamoorthy (1983) list, among others, the following adverse impacts of mercury in biota: inhibition of enzymes and protein synthesis in the liver, kidney, and brain; structural alterations of fish epidermal mucus; reduction of sperm viability; reduction of olfactory response; reduction of vision and respiration; decreased ability to osmoregulate; morphological changes in the gills; and, adverse effects on pancreatic tissue. Additionally, the WHO (1976) reported irreversible neurological damage to animals attributable to mercury toxicity.

Probably the most celebrated case of mercury contamination is that of Minamata Bay in Japan during the 1950's. In this case, mercury was discharged into the bay for over 30 years. Once in solution, mercury entered the food chain including, but not limited to, fish and shellfish, mammals, and birds. Large number of dead fish and other wildlife were seen floating on the sea surface and are thought to have died from exposure to mercury (Doi et al, 1984). Doi (1984) found that fish-eating birds had the highest concentration of mercury

when compared to herbivorous waterfowl, leading him to establish a relationship between the particular food item and the mercury concentrations in animals.

Eisler (1987a) reports a range between 0.1 and 2.0 ug/l (ppb) total mercury in water as lethal to aquatic organisms. He concluded that maximum total mercury concentrations should not exceed 100 ug/kg (ppb) fresh weight in bird's prey and 1,100 ug/kg (ppb) in small mammals' prey, and that mercury tissue concentrations exceeding 1,100 ug/kg (ppb) fresh weight should be regarded as evidence of an environmental mercury problem. Kent and Johnson (1979) report mercury levels in uncontaminated fish of less than 0.2 ppm. Mercury levels in Connecticut River fish do not appear to be of concern at this time.

TABLE VII - WEIGHTED MERCURY CONCENTRATIONS IN FISHES

SPECIES	CONC. (UG/G) WW	LOCATION	REFERENCE
SMALLMOUTH BASS	ND- 0.250	PENNSYLVANIA	ROMPALA et al, 1984
	0.31-1.17	NEW YORK	ARMSTRONG AND SLOAN, 1980
	0.330	PENOBSCOT RV.,ME	NCBP, 1984
	0.204	BELOW ASHUELOT RV.	PRESENT STUDY
	0.113	BRATTLEBORO	
	0.060	CLAREMONT	
	0.077	WEST LEBANON	
	0.168	HANOVER	
WHITE SUCKER	ND- 0.430	PENNSYLVANIA	ROMPALA et al, 1984
	0.18-0.54	NEW YORK	ARMSTRONG AND SLOAN, 1980
	0.180	PENOBSCOT RV.,ME	NCBP, 1984
	0.210	MERRIMACK RV.,MA	
	0.060	CLAREMONT	PRESENT STUDY
YELLOW PERCH	0.220	CONNECTICUT RV.,CT	NCBP, 1984
	0.160	ANDROSCOGGIN RV.,ME	
	0.088	BRATTLEBORO	PRESENT STUDY
	0.112	CLAREMONT	
	0.086	WEST LEBANON	
	0.174	HANOVER	
WHITE PERCH	0.710	NEW YORK	ROMPALA et al, 1984
	0.060	SUSQUEHANNA RV.,MD	NCBP, 1984
	0.096	BELOW ASHUELOT RV.	PRESENT STUDY
	0.091	BRATTLEBORO	
	0.136	WEST LEBANON	
WALLEYE	0.100	PENNSYLVANIA	ROMPALA et al, 1984
	0.600	NEW YORK	ARMSTRONG AND SLOAN, 1980
	0.193	HANOVER	PRESENT STUDY
CHAIN PICKEREL	0.159	BELOW ASHUELOT RV.	PRESENT STUDY

1.4.5 - PCBs

PCBs (Polychlorinated biphenyls) are a group of organic compounds that were first produced commercially in the U.S. in 1929 (Moore and Ramamoorthy, 1984). Total PCBs production in the U.S. has been estimated at 600,000 metric tons from 1929 until they were banned in 1979 (Weaver, 1984).

PCB distribution is now worldwide due to accidental spills and improper disposal and, because they are somewhat volatile, and are disseminated in the atmosphere. PCB-containing materials such as transformers and capacitors still in service present a potential future source of discharge into the environment.

PCBs are found in the atmosphere, rain water, rivers, lakes, oceans, marine and terrestrial mammals, birds, bird eggs, fish, mollusks, marine plants, and even Antarctica biota (Gustafson, 1970). PCBs are hydrophobic and persistent in the environment, with a tendency to accumulate in sediments (by adsorption) where they become available to benthic organisms (Larsson, 1986). Biodegradation appears to be the dominant fate process for those biphenyls with four chlorine atoms or less (Callahan et al,1979). Biphenyls with five chlorine atoms or more are more resistant to biodegradation and thus are more persistent in the environment (Callahan et al, 1979; EPA, 1980b). According to EPA (1980b), PCBs can be transformed to PCDFs (Polychlorinated dibenzofurans) in the environment by either heat, light, or metals and metal salts. PCDFs are potentially more toxic than PCBs. Photodegradation appears to play the most important role in the breakdown of the more highly chlorinated PCBs, though it is a slow process according to Callahan et al (1979).

Volatilization, while important, is depressed when organic solids are present and sorption by solids with organic content is strong. PCBs are bioaccumulated and biotransformation is important in the destruction of those with fewer than four chlorines per molecule.

Due to high lipophilicity, PCBs can bioaccumulate and biomagnify within the food chain (Eisler, 1986b; Metcalf et al, 1975) at concentrations in water that are often below the detection limits (EPA, 1980b; Gooch and Hamdy, 1982). Because the lipid content of an organism affects the amount of PCB it can bioaccumulate, seasonal variation in uptake is expected because body fat varies seasonally.

The primary active site for PCB metabolism is the liver (Moore and Ramamoorthy, 1984) and PCB residues are generally lower in the muscle tissue than in the viscera (Moore and Ramamoorthy, 1984; Farrington et al, 1986). Juvenile and immature invertebrates are more sensitive to PCBs than adults, and toxicity varies inversely with chlorine content of the biphenyls (Eisler, 1986b). PCB uptake is higher with increasing chlorination (Ernst, 1984). Females have faster PCB depuration rates than males because egg laying is an important route of PCB elimination (Eisler, 1986b). Decreased growth of individuals and changes in community structure of aquatic organisms due to PCBs have been well documented (Eisler, 1986b; EPA, 1980). Fish accumulate PCBs to relatively high levels and when exposure ceases they are not quickly eliminated (Rompala et al, 1984). High PCB residues in fish are also a potential hazard to fish-eating wildlife such as mink, otter, and fisher, among others.

The National Academy of Science has set a total PCB level of 0.5 ppm as the maximum level for the protection of fish-eating wildlife (Rompala et al, 1984). This level is exceeded in smallmouth bass from Brattleboro, yellow perch and white sucker from Claremont, and smallmouth bass, chain pickerel, and white perch from below the Ashuelot River.

TABLE VIII - WEIGHTED PCBs CONCENTRATIONS IN FISHES

SPECIES	CONC. (UG/G) WW	LOCATION	REFERENCE
SMALLMOUTH BASS	ND- 2.990	PENNSYLVANIA	ROMPALA et al, 1984
	0.24-3.10	NEW YORK	ARMSTRONG AND SLOAN, 1980
	0.46-2.80	MERRIMACK RV.,NH & MA	UNPUBLISHED
	2.100	CONNECTICUT RV.,CT	NCBP,1984
	0.704	BELOW ASHUELOT RV.	PRESENT STUDY
	0.593	BRATTLEBORO	
	0.239	CLAREMONT	
	0.276	WEST LEBANON	
	0.346	HANOVER	
WHITE SUCKER	ND- 6.360	PENNSYLVANIA	ROMPALA et al, 1984
	0.57-1.04	NEW YORK	ARMSTRONG AND SLOAN
	0.70-4.10	MERRIMACK RV.,NH & MA	UNPUBLISHED
	0.03-1.64	CONNECTICUT RV.,CT	
	2.500	MERRIMACK RV.,MA	NCBP, 1984
	0.980	CLAREMONT	PRESENT STUDY
YELLOW PERCH	0.31-4.81	NEW YORK	ARMSTRONG AND SLOAN, 1980
	0.09-1.39	MERRIMACK RV.,NH & MA	UNPUBLISHED
	0.75-1.39	CONNECTICUT RV.,CT & MA	
	1.500	CONNECTICUT RV.,CT	NCBP, 1984
	0.371	BRATTLEBORO	PRESENT STUDY
	0.658	CLAREMONT	
	0.114	WEST LABANON	
	0.306	HANOVER	
WHITE PERCH	4.99-85.4	NEW YORK	ARMSTRONG AND SLOAN, 1980
	0.900	SUSQUEHANNA RV.,MD	NCBP, 1984
	1.876	BELOW ASHUELOT RV.	PRESENT STUDY
	0.488	BRATTLEBORO	
	0.166	WEST LABANON	
WALLEYE	0.63-2.78	NEW YORK	ARMSTRONG AND SLOAN, 1980
	0.120	PENNSYLVANIA	ROMPALA et al,1984
	0.333	HANOVER	PRESENT STUDY
CHAIN PICKEREL	0.779	BELOW ASHUELOT RV.	PRESENT STUDY

1.4.6 - PAHs

PAHs (Polycyclic aromatic hydrocarbons) are ubiquitous to the environment. They may result from natural sources, such as volcanic activity and forest fires (Eisler, 1987b), and from anthropogenic sources, with the highest concentrations occurring around industrialized areas (Callahan et al, 1979; Moore and Ramamoorthy, 1984).

Municipal and industrial effluents, atmospheric fallout, fly ash precipitation, road and urban runoff, and leaching of contaminated soils are pathways for PAHs to reach surface waters (Eisler, 1987; Moore and Ramamoorthy, 1984). As an example, in Narragansett Bay, Rhode Island, urban runoff accounts for 36% of total PAHs (Hoffman et al, 1984). Different oils have different PAH concentrations (Graf and Winter, 1968) depending on the distillation method and the degree to which it is refined (Moore and Ramamoorthy, 1984).

Chemically, PAHs are composed of two or more benzene rings fused together in different arrangements, which affects their physical and chemical properties, as well as their fate and interaction with biological systems according to Moore and Ramamoorthy (1984). They also pointed out that many PAHs have been identified, but few are produced commercially, and that the most common are: naphthalene, acenaphthene, fluorene, and phenanthrene.

PAHs have high molecular weight and low polarity. As the former increases, so does lipophilicity, while aqueous solubility decreases. However, water solubility may be increased by the presence of other substances such as anionic compounds (Moore and Ramamoorthy, 1984).

PAHs are sorbed to particulate matter and can accumulate to high concentrations in sediments. In general, even though PAH concentrations are low in the water column due to low solubilities, aquatic organisms may have concentrations several orders of magnitude higher. The concentrations of PAHs in sediments is frequently higher than in many biota because some organisms can metabolize the parent PAHs, often to more toxic compounds (Moore and Ramamoorthy, 1984).

Photolysis, microbial- and photo-oxidation, sedimentation, and to a lesser degree, volatilization, contribute to PAH depletion in water. PAHs may be more persistent in eutrophic than in oligotrophic waters since photolysis would be reduced in the former (Callahan et al, 1979). Microorganisms in soil, sewage, and water are capable of degrading PAHs (Moore and Ramamoorthy, 1984).

Benzo(a)pyrene, fluoranthene, pyrene, and anthracene are the PAHs most frequently found in sediments. Of these, benzo(a)pyrene has been classified as a carcinogen (EPA, 1980d; Moore and Ramamoorthy, 1984).

Sorption is considered the most important transport process via sediments and suspended solids. Bioaccumulation is a short term process for PAHs with four aromatic rings or more which are slowly metabolized and long-term partitioning into biota is not significant. PAHs with four or more aromatic rings are degraded by microbes and metabolized by multicellular organisms. Biodegradation is considered the ultimate fate process.

Although PAHs are not acutely toxic to living organisms (Moore and Ramamoorthy, 1984), several of them are among the most potent carcinogens known to exist (Eisler, 1987b). Some of their metabolites appear to be mutagens, teratogens, and carcinogens (Heidelberger, 1976; Moore and Ramamoorthy, 1984). Some PAHs have produced skin tumors in mice, rabbits (Dipple, 1985) and fish (Black, 1984). Although low molecular weight PAHs (2-3 ring aromatics) have relatively high acute toxicity compared to heavier PAHs, their chronic toxicity is much lower. Higher molecular weight PAHs have proven to be carcinogens (Eisler, 1987b).

Detectable PAH levels have been recorded for invertebrates in areas as remote as the Antarctic (Platt and Mackie, 1981). In fresh water and marine fish, PAH concentrations are generally low, except in localized problem areas. Uptake by fish is rapid and increases as concentration in the environment increases, but it is a short term accumulation, at least of parent compounds, because PAHs are metabolized and/or excreted (Moore and Ramamoorthy, 1984). In mammals, the liver metabolizes PAHs, while in other vertebrates and invertebrates, the liver and other organs accomplish this function. Fish and crustaceans possess enzymes that metabolize PAHs, but mollusks are unable to break down these compounds (Jackin and Lake, 1978; Varanasi et al, 1985). Moles et al, (1981) reported reduction of growth and fecundity in some aquatic species exposed to PAHs. Moore and Ramamoorthy (1984) reported PAH-related behavioral abnormalities in fish, such as loss of equilibrium, avoidance, and physiological effects such as glycogen depletion, development of gonadal tumors, and stomach and intestine abnormalities.

PAH bioaccumulation in aquatic systems does not appear to be a dominant fate process except on a short term basis since they are absorbed and eliminated rapidly by most vertebrates (Callahan et al, 1979). However, some invertebrates, particularly mollusks, have difficulty eliminating PAHs. Bioaccumulation by benthic organisms and biodegradation is slow in aquatic systems but the latter is an important process in chronically affected systems (Callahan et al, 1979).

We did not include a table for total PAHs because the available values in the literature are compound specific.

1.4.7 - DDT, DDE, and DDD

DDT (Dichloro-diphenyl-trichloroethane), is a chlorinated pesticide first synthesized in 1874 in Germany that has been widely used throughout the world since 1939. Although DDT has been banned in the U.S. since 1969 (EPA, 1980, Moore and Ramamoorthy, 1984), DDE (Dichloro-diphenyl-dichloroethylene) and DDD (Dichloro-diphenyl-dichloroethane) remain persistent metabolites of DDT (Menzie, 1980).

DDT, DDE, and DDD are very persistent in the environment and volatilization, sorption to sediments, and bioaccumulation are dominant fate processes according to Callahan et al (1979). They also suggest that their ultimate transformation in the aquatic environment is by biotransformation. They are highly lipophilic and because of their volatility, susceptible to large-scale transport.

As hydrophobic organic compounds, DDT and its metabolites tend to bioaccumulate differentially in the lipids of biota (Armstrong and Sloan,1980). Plants absorb them rapidly and efficiently and they are readily bioaccumulated by fish (Suns et al, 1981). Factors affecting chlorinated pesticides uptake by fish include: species (salmonids appear to be more sensitive than centrarchids according to Brown, 1978), lipid levels, age, size, metabolic rate, reproduction, and feeding conditions (Moore and Ramamoorthy, 1984). Chlorinated insecticides inhibit normal function of the gills in fish, thus affecting osmoregulation, hemoglobin and plasma protein levels, and adversely affecting the spleen, liver, and kidney (Poels et al, 1980, Brown, 1978). Enzyme inhibition and metabolic changes often have obvious

external effects such as retardation of fin regeneration, development of goiters, and change in the discrimination ability and temperature selection thereby directly affecting fish survival (Moore and Ramamoorthy, 1984).

DDD concentrations of fish in water with 14 to 20 ppb have ranged between 5 and 20 ppm, with visceral levels exceeding 2000 ppm (Callahan et al, 1979). Sublethal effects of all organochlorine insecticides are of concern because of their toxicity and persistence.

The major form of DDT-compounds detected was as DDE, which is expected considering the mechanism of uptake and metabolism of DDT by biota (Brooks, 1974).

The National Academy of Science and National Academy of Engineering (NAS-NAE) recommend that whole body residues should not exceed 1.0 ug/g ww total DDT (including DDE and DDD)(Winger et al, 1984). This value was not exceeded in any of our samples.

TABLE IX - WEIGHTED DDT (TOTAL) CONCENTRATIONS IN FISHES

SPECIES	CONC. (UG/G) WW	LOCATION	REFERENCE
SMALLMOUTH BASS	0.07-0.41	PENNSYLVANIA	ROMPALA et al, 1984
	0.05-0.21	MERRIMACK RV.,NH & MA	UNPUBLISHED
	0.900	DELAWARE RV.,NJ & PA	NCBP, 1984
	0.088	BELOW ASHUELOT RV.	PRESENT STUDY
	0.080	BRATTLEBORO	
	0.037	CLAREMONT	
	0.019	WEST LEBANON	
	0.072	HANOVER	
WHITE SUCKER	ND - 1.46	PENNSYLVANIA	ROMPALA et al, 1984
	0.07-0.19	MERRIMACK RV.,NH & MA	UNPUBLISHED
	0.07-0.14	CONNECTICUT RV.,MA & CT	
	0.30-0.41	DELAWARE RV.,NJ & PA	NCBP, 1984
	0.13-0.16	RARITAN RV.,NJ	
	0.018	CLAREMONT	PRESENT STUDY
YELLOW PERCH	0.02-0.09	MERRIMACK RV.,NH & MA	UNPUBLISHED
	0.08-0.10	CONNECTICUT RV.,MA & CT	
	0.170	CONNECTICUT RV.,CT	NCBP, 1984
	0.062	BRATTLEBORO	PRESENT STUDY
	0.128	CLAREMONT	
	0.064	HANOVER	
WHITE PERCH	0.120	SUSQUEHANNA RV.,MD	NCBP, 1984
	0.204	BELOW ASHUELOT RV.	PRESENT STUDY
	0.083	BRATTLEBORO	
	0.048	WEST LEBANON	
WALLEYE	0.010	PENNSYLVANIA	ROMPALA et al, 1984
	0.500	HANOVER	PRESENT STUDY
CHAIN PICKEREL	0.132	BELOW ASHUELOT RV.	PRESENT STUDY

SECTION 2 - WILDLIFE CONCLUSIONS AND RECOMMENDATIONS

The following comments should be viewed keeping in mind that this work was meant as a preliminary survey. We hope that this is the beginning of a cooperative effort by the various agencies involved and that sources of potential problems can be identified and prompt corrective measures taken. A biennial sampling program is desirable and in the interest of the State of New Hampshire and other states within the river basin.

According to Sloan (1983), cadmium levels in the tissue higher than 0.1 ppm results in adverse reproductive effects. Levels higher than this value were observed in two species at West Lebanon (See Table III).

Eisler (1986a) gives a value of 0.8 ppm as presumptive evidence of chromium contamination. This level was exceeded in smallmouth bass from stations 4 (Brattleboro) and 5 (below the Ashuelot River); yellow perch from station 3 (Claremont); white perch from station 5 (below the Ashuelot River); white sucker from station 3 (Claremont); and chain pickerel from station 5 (below the Ashuelot River). From the available information it is not possible to determine the effects these levels are having in the biota.

Lead does not appear to represent a problem in the samples taken since none exceeded the 1 ppm value reported by Rompala et al (1984) as representing a contaminant problem.

Mercury levels of 1.1 ppm in tissue should be regarded as evidence of an environmental problem according to Eisler (1987a). None of the samples taken for this work reach this value.

The National Academy of Science has set 0.5 ppm total PCBs as the maximum concentration in fish for the protection of fish-eating wildlife. This value was exceeded in samples taken from Claremont, Brattleboro, and below the Ashuelot River.

PAHs do not appear to be a problem in biota taken from the Connecticut River. Total DDT values should not exceed 1.0 ppm in tissue according to the National Academy of Science and the National Academy of Engineering. Samples in this survey do not exceed this value.

In summary, cadmium exceeds acceptable levels at West Lebanon; PCBs exceed acceptable levels at Claremont, Brattleboro, and below the Ashuelot River; and chromium exceeds acceptable levels at all stations.

It is recommended that further work be done which would involve taking more fish samples and sediments as well. Individual rather than composite samples are more desirable.

SECTION 3 - HUMAN HEALTH RISK ASSESSMENT

3.1 - INTRODUCTION

The DPHS performed a human health risk assessment to estimate the potential risks to human consumers of Connecticut River fish. This focuses on the potential effects of chronic exposure to those chemical contaminants selectively surveyed in this study.

The initial step was to perform a toxicity assessment using data presented in Section 1. The toxicity assessment provides toxicological information for the contaminants detected in Connecticut River fish. Brief summaries are provided, concentrating on toxicological properties associated with exposure via oral ingestion. A dose response assessment is presented with pertinent criteria necessary to evaluate which dose would be expected to pose a public health concern.

The exposure assessment characterizes the populations exposed to contaminants in fish and delineates human consumption rates for fish. Consumption rates are then integrated with contaminant concentrations to estimate exposure levels.

In the risk characterization step, the probability and extent of adverse health effects associated with consumption of fish from the Connecticut River are estimated. In order to determine whether the levels of contaminants detected in fish would pose unacceptable risks to human health if ingested, the DPHS compared the estimated exposure levels to maximum acceptable exposure levels (see Dose-Response section for a description of how maximum acceptable exposure levels are determined).

3.2 - TOXICITY ASSESSMENT

3.2.1 - DOSE-RESPONSE ASSESSMENT

The dose-response assessment is a description of the relationship between the dose of a chemical and the incidence of the adverse effect in a population.

Various criteria can be used in a dose-response assessment to determine acceptable oral exposure levels. For evaluating carcinogenic risks, we have used cancer potency factors (CPFs) which relate the dose of a carcinogen to an expected increased risk of cancer. For evaluating those health risks other than cancer, we have compared estimated doses to reference dose values (RfDs) or permissible tolerable daily intakes (PTDIs). These criteria are discussed in more detail below. Table X provides the specific values for these criteria for each chemical evaluated in this report.

Another value which the DPHS uses to help determine a maximum acceptable level for contamination in fish are action or tolerance levels, established by the U.S. Food and Drug Administration (FDA). As opposed to the other criteria (mentioned above) which represent allowable exposure levels, the FDA action level refers to an allowable concentration in the fish themselves (Table X).

Weight of Evidence for Human Carcinogenicity

The process which the U.S. EPA uses to determine the strength of evidence that a chemical causes cancer in humans is primarily based on findings which are reported from animal and human studies. The U.S. EPA has developed a set of criteria which are used to assign a chemical to one of six different "weight of evidence" categories, which are listed in Appendix B. Chemicals that are assigned to the highest weight of evidence category are designated as Group A

- human carcinogens. Chemicals designated by the U.S. EPA as Group A carcinogens must have sufficient evidence from human epidemiologic studies which support a causal association between exposure to that chemical and cancer (U.S. EPA, 1986a).

However, the chemicals surveyed in this study have all been assigned by the U.S. EPA either a Group B2 or Group D classification for oral exposure (Table 1). Group B2 (probable human carcinogens) include those chemicals for which there is sufficient evidence from animal studies, but for which there is either "inadequate evidence" or "no data" from human epidemiologic studies. Group D carcinogens (not classifiable as to human carcinogenicity) generally have either inadequate evidence of carcinogenicity or else no data are available to make such a determination (U.S. EPA, 1986a)

Cancer Potency Factors (CPFs)

EPA calculates cancer potencies using a linearized multistage model for low-dose extrapolation. This model derives a plausible upper limit (upper 95% confidence limit) of carcinogenic risk for an exposed population. Cancer potency factors are calculated by assuming daily exposure for 70 years, and this value is expressed in units of $(mg/kg/day)^{-1}$.

When the CPF is multiplied by the estimated exposure level, this results in an estimated "upper bound" population cancer risk level. The equation used to calculate the upper-bound cancer risk level is as follows:

$$\text{Upper-Bound Cancer Risk Level} = \text{Cancer Potency Factor } (mg/kg/day)^{-1} \times \text{Exposure Level } (mg/kg/day)$$

A population cancer risk is reported as the number of excess cancers resulting from exposure to a given number of people. For example, a cancer risk of 1×10^{-6} represents one excess cancer in one million people exposed.

Reference Dose (RfD)

The reference dose (RfD) can be used to estimate a level of oral exposure at or below which no adverse noncarcinogenic effect is expected to occur. The RfD is an estimate of a daily oral exposure to the human population that is likely to be without appreciable risk of deleterious effects during a lifetime. The RfD is derived from an observed threshold dose in a chronic animal bioassay, and by applying an uncertainty factor, which usually ranges from 1 to 1000. The uncertainty factors are incorporated to account for such factors as extrapolation from animal data to humans, variability in toxic susceptibilities in human populations, and the lack of long-term exposure data. RfDs are expressed in units of mg/kg/day.

FDA Action and Tolerance Levels

The FDA has established action and tolerance levels for contaminants in food, such as fish (U.S. FDA, 1982). Action and tolerance levels, expressed in units of ug contaminant per gram of fish (or ppm), are intended to represent a limit at or above which the FDA will take legal action to remove adulterated products from the market. Tolerance levels are similar to action levels, except that they have undergone a formal rulemaking process and, therefore, have the force of the law (U.S. FDA, 1988). Action levels are general statements of policy that are intended for interim periods and can be instituted and changed more quickly than tolerances. It is noted that action/tolerance levels are predicated not only on safety but also on factors such as economic impact of the food industry in complying with the established levels (U.S. EPA/FDA, 1988). Therefore, these values are not entirely health based, and a certain level of risk may exist from consumption of fish which are contaminated with a chemical even below this standard.

3.2.2 - HAZARD IDENTIFICATION

The following text briefly describes the toxicity of those compounds which were analyzed in fish sampled from the Connecticut River. Since this study focuses on health risks resulting from the ingestion of fish, these toxicity assessments will emphasize health effects from chronic oral exposure.

3.2.2.1 - Inorganic Compounds

Cadmium

Cadmium (Cd) ingested in food or water is poorly absorbed from the gastrointestinal (GI) tract (1 to 6%) in both humans and animals, and once absorbed into the body, it tends to accumulate in the kidney and liver (ATSDR, 1987a). The most important adverse health effect resulting from chronic Cd exposure is renal tubular damage, although such exposures reportedly seldom lead to end-stage renal disease (U.S. EPA, 1980). Based on toxic effects to the kidney, the U.S. EPA has reported an oral risk reference dose (RfD) for Cd, which is equal to 5×10^{-4} mg/kg/day (see Table X).

Unlike inhalation, there is no strong evidence to indicate that Cd is carcinogenic when ingested, and the U.S. EPA has classified this compound as a group D carcinogen (not classifiable as to human carcinogenicity) for this route of exposure.

Chromium

Chromium (Cr) can occur in different valence states, such as chromium III (Cr III) or chromium VI (Cr VI), and the latter is considered responsible for the majority of health problems associated with total Cr exposure. While both trivalent and hexavalent Cr are found in nature, Cr(III) is reported as the predominant form. Chromium in food is also reported to be present mostly in the trivalent form (ATSDR, 1987c).

It is estimated that no more than 1% of Cr in foods is likely to be absorbed from the GI tract (Carson, 1987). The kidney appears to be the main target organ for acute Cr toxicity, with effects occurring at doses of 1-2 mg Cr (VI)/kg body weight (U.S. EPA, 1984a). The U.S. EPA has calculated a risk reference dose (RfD) equal to 5×10^{-3} mg/kg/day for Cr VI (see Table X). No oral RfD has been reported for trivalent chromium.

Much of the information on the effects of Cr VI to humans is obtained from occupational exposures for which the predominant health effects are on the respiratory system and skin (U.S. EPA, 1984a). The U.S. EPA has classified Cr VI as a group A carcinogen by inhalation, but there is no evidence it is carcinogenic by ingestion. Chromium III has been assigned an EPA classification of group D (not classified as to human carcinogenicity) by oral ingestion.

Lead

In young children, it is estimated that approximately forty to fifty percent of total ingested lead (Pb) is absorbed into the body as compared with eight to fifteen percent in adults (ATSDR, 1988). Excessive levels of Pb in the human body can cause serious damage to the brain, kidneys, nervous system, and red blood cells (U.S. EPA, 1987c). The endpoints most sensitive to low-level exposure to lead are neurobehavioral deficits and growth retardation in young children, and hypertension in middle-aged men. The most serious effects to the central nervous system observed in children at low levels of exposure include hyperactivity and decreased IQ scores. Young children, infants and fetuses are considered to be more vulnerable to lead poisoning than adults. A Health Advisory of 20 ug/day has been established by U.S. EPA, which takes into account protection of young infants (U.S. EPA, 1985b). Based on this Health Advisory the DPHS has derived an adjusted oral RfD, equal to 2.20×10^{-3} mg/kg/day (see Table X).

The U.S. EPA has classified Pb as a group B-2 (probable human) carcinogen, based on twelve studies involving rats and mice having associated kidney tumor formation with high doses of Pb salts (U.S. EPA, 1985b). However, no cancer potency factor (CPF) has been estimated for Pb by the U.S. EPA Carcinogen Assessment Group (CAG).

Mercury

A survey that was conducted by the U.S. FDA indicates that 99.5% of mercury ingestion arises from the meat, fish and poultry food group (U.S. DHEW, 1979). Since methyl mercury is the predominant form in fish tissue, we have focused this toxicity summary on methyl mercury.

Methyl mercury acts by selectively damaging the central nervous system in humans. Severe exposures result in destruction of neuronal cells in the central nervous system that are involved with sensory and coordination functions, and can lead to conditions of deafness, slurred speech, or mental derangement (U.S. EPA, 1984d). Lower exposure levels can result in non-specific symptoms such as paresthesia (abnormal sensation, as burning, prickling, etc.), malaise (vague feeling of bodily discomfort), and blurred vision. Prenatal life is considered to be especially vulnerable to methyl mercury, as severe brain damage has been observed in infants whose mothers have ingested this compound during pregnancy. Based on toxic effects to the central nervous system, the U.S. EPA has established an oral RfD for methyl mercury, equal to 3.0×10^{-4} mg/kg/day (see Table X).

While the FDA has developed an Action Level in fish equal to 1 ppm, some states, such as Michigan, have adopted their own action level for mercury concentration in fish at more stringent levels (0.5 ppm; Humphrey and Hesse, 1986).

3.2.2.2 - Organic Compounds

DDT, DDE and DDD

Both DDT and DDE are absorbed from the gastrointestinal tract with high efficiency. Once absorbed, both DDT and DDE become distributed into the fatty tissue (U.S. EPA, 1984b; U.S. EPA, 1980b).

The most commonly reported adverse effect from chronic exposure to DDT in experimental animals is liver toxicity. Although there is no strong evidence that DDT is teratogenic (i.e., does not exhibit the ability to produce physical defects in the developing embryo), oral exposures to several species of experimental animals have yielded decreased reproductive capacity (U.S. EPA, 1984b). Based on toxic effects to the liver the U.S. EPA has reported an oral RfD for DDT, equal to 5×10^{-4} mg/kg/day (see Table X).

Studies of occupational exposure to DDT have not found any increased incidences of cancer, although these studies may have been too limited in duration and scope (IARC, 1974). Animal studies in mice have shown DDT, DDE and DDD to produce liver tumors upon oral exposure (U.S. EPA, 1986b). Lifetime oral administration of DDD to this same species of mice has produced a marked increase in lung tumors (U.S. EPA, 1980a). Accordingly, the U.S. EPA has classified each of these chemicals as group B-2 carcinogens by oral exposure (Table X; U.S. EPA, 1986b). The EPA has developed cancer potency factors for DDT, DDE and DDD, which are equal to 0.34, 0.34, and 0.25 $(mg/kg/day)^{-1}$, respectively.

Polychlorinated Biphenyls

Human exposure to polychlorinated biphenyls (PCBs) has resulted largely from consumption of contaminated food, but also from inhalation and dermal exposure in the workplace.

Studies on the absorption of PCBs following oral exposure indicate that GI absorption of most congeners is greater than ninety percent (ATSDR, 1987c). Ingestion of PCBs by laboratory rats has resulted in the highest concentrations in the adipose tissue, followed by the mammary glands, kidney, liver and lung (McCormack et al, 1979). Studies in both experimental animals and humans indicate that once PCBs are absorbed from the GI tract, they can be distributed into the breast milk of exposed mothers (Curley et al, 1973; Schwartz et al, 1983).

Accidental human ingestion of PCBs has resulted in various health effects, including acneform eruptions and abnormal hepatic function (U.S. EPA, 1985c). High serum PCB levels among pregnant women have been associated with increased abortions (Bercovici et al, 1983) and premature deliveries (Wassermann et al, 1982). Oral exposures to experimental animals has resulted in liver toxicity, skin lesions and indications of altered immune responses. There is no available oral RfD that has been reported for PCBs.

Experimental animals exposed to PCBs have developed an increased incidence of liver tumors (U.S. EPA, 1985c). The U.S. EPA has classified PCBs as a group B-2 (probable human) carcinogen.

Polynuclear Aromatic Hydrocarbons (PAHs)

PAHs are highly lipid-soluble and it has been proposed that they are readily absorbed from the GI tract. Upon reaching the bloodstream, PAHs are rapidly distributed to most internal organs with extensive localization in the fat (U.S. EPA, 1980c).

PAHs are of concern to human health because they may cause cancer. The U.S. EPA has classified benzo(a)pyrene (B[a]P), which is just one of the many PAH constituents, as a group B-2 carcinogen by oral exposure (U.S. EPA, 1987b). Ingestion of B[a]P in the diet by experimental mice has resulted in the induction of stomach tumors. Regarding non-carcinogenic toxicity, PAHs may have an effect on the immune and cardiovascular systems. PAHs reportedly affect tissues which exhibit rapid proliferation, such as the intestinal epithelium, bone marrow, lymphoid organs, and testis (U.S. EPA, 1980c). No oral RfD has been reported by U.S. EPA for PAHs.

3.3 - EXPOSURE ASSESSMENT

The exposure assessment is the process of characterizing the population of concern, determining relevant consumption rates, and estimating a subsequent exposure dose. The exposure route of concern for this health risk assessment is ingestion of freshwater fish from the Connecticut River.

3.3.1 - POPULATION OF CONCERN

There are no angler creel census data available for the study reach of the Connecticut River at this time. There has been no commercial harvest reported along the New Hampshire/Vermont reach of the Connecticut River. The population of concern for this risk assessment is assumed to be recreational fishermen and their families.

3.3.2 - CONSUMPTION RATES

Since specific information on catch and consumption patterns are not available for the study reach of the Connecticut River, and most likely vary over time, estimated average freshwater fish consumption rates were used. The U.S. EPA uses an estimate of 6.5 grams/day in their process of developing water quality criteria to approximate an average level of fish and shellfish consumption (PTI Environmental Services, 1987).

However, there have been reports in the literature which indicate that the average amount of fish consumption has increased over the past decade, suggesting that the consumption rate of 6.5 g/day may underestimate the actual consumption rate today (Zar, 1988). In a recent risk assessment conducted by the U.S. EPA (1988), which investigated exposure to dioxin from consumption of fish from the Tittabawassee River in Michigan, an average consumption rate of 7.8 g/day was used to represent the "general consumer" (U.S. EPA, 1988).

This average consumption rate (7.8 g/day) is based on a more recent study conducted by the U.S. Department of Agriculture, which sampled 37,874 individuals throughout the United States for a three day period (USDA, 1982). The survey's results indicated that 14.5% of the people sampled consumed "finfish other than canned, dried, or raw" with an average intake of 54 g/day. When averaged over the entire population sampled, an overall average consumption rate of 7.8 g/person/day was obtained. This consumption rate of 7.8 g/day will be used in this assessment to represent the general consumer of fish from the Connecticut River.

In the same risk assessment conducted by the U.S. EPA of the Tittabawassee River mentioned above, a consumption rate of 48 g/day was used by the U.S. EPA to represent fishermen and their families (U.S. EPA, 1988). This consumption rate (48 g/day) was based on a survey of a large population of active "sports fishermen," which reported a median consumption rate of 38.5 lbs/year, or 48 g/day (Humphrey, 1983). This consumption rate will be used in this risk assessment to approximate the median daily consumption by avid sports fishermen (and their families) who fish the Connecticut River.

3.3.3 - ESTIMATED EXPOSURE LEVELS

The equation used to calculate exposure levels is as follows:

$$\text{Exposure Level (mg/kg/day)} = \frac{\text{Concentration in Fish (ug/g)} \times \frac{1\text{ mg}}{1000\text{ ug}} \times \text{Consumption Rate (g/day)} \times \text{Proportion of Exposure Duration (yrs/yrs)}}{\text{Body Weight (kg)}}$$

Exposure levels were determined by considering the following factors: contaminant concentrations in fish, consumption rates, exposure duration, and body weight. Only the concentrations of contaminants in the fish, themselves, can be measured with precision. However, there is still some uncertainty as to whether the fish sampled are representatives of other fish from the same river. The other exposure factors, such as consumption patterns, are merely estimates. As a worst case scenario, in this health assessment it is assumed that the total dietary intake of fish which these people consume consists of those taken from the Connecticut River.

Chemical concentrations were measured in both fillet (skin off)- and carcass-composites. The fillet (often with the skin left on) is probably the most representative portion of the fish which humans generally consume. Since many people consume fillets with the skin on, exposure based on concentrations of contaminants in fillets with the skin off may underestimate exposure for certain chemicals. Specifically, lipid soluble contaminants (e.g., PCBs and DDT) are found at higher concentrations in fatty tissue, such as skin, than in the meat of the fish. Therefore, cooking or eating fish with the skin on would result in a higher exposure to lipid soluble contaminants.

It is also recognized that exposure levels for lipid soluble contaminants, calculated based on consumption of the carcass, will probably result in an over-estimate of the true exposure. Therefore, exposure levels were calculated separately for both fillet and carcass consumption, realizing that

the true level of exposure probably falls somewhere between the two.

For each chemical, an overall range of estimated exposure levels to the general fish consumer (assuming 7.8 g/day) is presented in Table XI. These ranges of exposure cross over all species and locations sampled in this study. To depict a worst-case scenario, a range of exposure levels were estimated assuming a high consumption rate equaling 48 g/day of fish (Table XI).

3.4 - RISK CHARACTERIZATION

In order to determine whether the levels of various chemicals detected in the fish species evaluated would pose an unacceptable risk upon ingestion, the DPHS has compared the range of estimated exposure levels presented in Table XI to levels which are considered acceptable (Table X).

3.4.1 - INORGANIC COMPOUNDS - Cd, Cr, Hg, Pb

While the various concentrations of inorganic chemicals were all within an acceptable range, some tended to be present at higher concentrations in certain locations. For example, cadmium levels in fish sampled at the W. Lebanon station were generally at least twice as high as those sampled from other locations. Levels of lead and mercury tended to be slightly higher in those fish sampled at the Hanover location compared with the others.

A review of the data did not indicate that there were any noticeable trends or differences for the levels of inorganic compounds between different species of fish, or for fillet versus carcass samples. Since inorganic chemicals are generally not expected to become concentrated to any great extent in fatty tissues, this may explain why there were no observed differences in concentrations between fillets and carcasses.

Except for lead, there is no sufficient weight-of-evidence to indicate that these inorganic chemicals would be carcinogenic to humans by oral ingestion. Since there is currently no available CPF that has been reported by the U.S. EPA for lead, it is not possible to estimate the cancer risk this chemical might pose from consumption of these fish. Therefore, the range of estimated exposure levels to each of the four inorganic chemicals is compared with noncarcinogenic criteria (RfDs, PTDIs) to determine if there is any indication of human health risk (Table X).

The estimated ranges of individual inorganic chemical exposures to people who consume either 7.8 g/day or 48 g/day (Table XI) were all found to be below those criteria which indicate a level of concern (Table X). However, it should be recognized that some level of carcinogenic risk may be associated with exposure to lead.

When two or more chemicals exert similar noncarcinogenic toxic effects to the same target organ, the DPHS will calculate a Hazard Index to assess their combined toxicity. A Hazard Index is calculated by taking the sum of the ratios of estimated exposure to the RfD, as follows:

$$\text{Hazard Index} = \frac{\text{Exposure}_i}{\text{RfD}_i}$$

Where, Exposure_i = exposure to the i^{th} toxicant
RfD = Oral RfD for the i^{th} toxicant

A Hazard Index is unitless. When a Hazard Index exceeds unity, this is considered to represent an unacceptable exposure.

Since cadmium and chromium both exert a toxic effect to the kidney, it is appropriate to evaluate their combined toxicity by computing a Hazard Index. Based on the lowest and the highest measured concentrations, the resulting ranges of hazard indexes are as follows:

Consumption Rate	Low	High
7.8 g/day	4.0×10^{-3}	7.4×10^{-2}
48 g/day	2.5×10^{-2}	4.5×10^{-1}

These ranges of Hazard Indexes for people who consume either 7.8 g/day or 48 g/day are below unity, which indicates that there would not be any anticipated adverse health effects posed as the result of combined exposure to cadmium and chromium from consumption of the fish.

Table X

Acceptable Oral Exposure Criteria for Various Compounds Detected in Connecticut River Fish

Compound	Oral EPA Carcinogen Classification	Oral CPF (mg/kg/day)[a]	U.S. EPA Oral Reference Dose (mg/kg/day)[b]	FAO/WHO PTDI[c] (mg/kg/day)	U.S. FDA Action Levels in Fish (ppm)[d]
Cadmium	D	_e	$5x10^{-4}$	$8.1x10^{-4}$-$1.0x10^{-3}$	-
Chromium	D	_e	$5x10^{-3}$	-	-
DDD	B-2	0.25	-	-	5 ppm[f]
DDE	B-2	0.34	-	-	5 ppm[f]
DDT	B-2	0.34	$5x10^{-4}$	-	5 ppm[f]
Lead	B-2	-	($2.2x10^{-3}$)[g]	$6.1x10^{-3}$	-
Mercury	D	-	($3.0x10^{-4}$)[h]	$6.1x10^{-4}$	1 ppm
PAHs[i]	B-2	11.5	-	-	-
PCBs	B-2	7.7	-	-	2 ppm

a. CPF stands for cancer potency factor. See dose-response section of this report for a description of this criterion.
b. A RfD is an estimate established by U.S. EPA of a daily exposure to the human population that is likely to be without appreciable risk of deleterious effects over a lifetime.
c. PTDI stands for provisional tolerable daily intake. These reported values have been converted from units of ug/day to mg/kg/day, assuming a human adult body weight of 70 kg.
d. This tolerance level refers to actual concentration of contaminant in the edible portion of the fish (excludes head, scales, viscera, and inedible bones). These levels, intended for interstate commerce, represent a limit at or above which FDA will take legal action to remove adulterated products from the market.
e. Although an inhalation-derived CPF is available for this chemical, this value is not considered suitable to use for oral exposure since the tumors occurred at the respiratory location.
f. For DDT and metabolites (collectively) including DDD and DDE.
g. DPHS has calculated this Oral RfD by converting from a U.S. EPA health advisory (HA) of 20 ug/day. Since this HA takes into account protection of young infants, this estimated RfD is calculated by assuming the weight of a child less than 1 year old is equal to 9 kg.
h. This RfD value is for methylmercury, which is the predominant form of total mercury present in edible fish.
i. These values are for benzo(a)pyrene, which is only one of the many chemical compounds which make up PAHs.

Table XI

Ranges of Inorganic Chemical Concentrations in Connecticut River Fish and Corresponding Estimated Exposure Levels

Chemical	Range of Concentration[a]	Estimated Exposure Levels[b] (mg/kg/day) General Consumer[c]	Active Sports[d]
CADMIUM			
Lowest Conc.	0.010	1.11×10^{-6}	6.86×10^{-6}
Species	S. Mouth Bass		
Location	Claremont		
Highest Conc.	0.180	2.01×10^{-5}	1.23×10^{-4}
Species	White Perch		
Location	W. Lebanon		
CHROMIUM			
Lowest Conc.	0.079	8.80×10^{-6}	5.42×10^{-5}
Species	S. Mouth Bass		
Location	Brattleboro		
Highest Conc.	1.500	1.67×10^{-4}	1.03×10^{-3}
Species	Y. & W. Perch		
Location	Brattleboro Below Ashuelot		
LEAD			
Lowest Conc.	0.039	4.35×10^{-6}	2.67×10^{-5}
Species	White Sucker		
Location	Claremont		
Highest Conc.	0.540	6.02×10^{-5}	3.70×10^{-4}
Species	S. Mouth Bass		
Location	Hanover		
MERCURY			
Lowest Conc.	0.060	6.69×10^{-6}	4.11×10^{-5}
Species	White Sucker & S. Mouth Bass		
Location	Claremont & Below Ashuelot		
Highest Conc.	0.290	3.23×10^{-5}	1.99×10^{-4}
Species	S. Mouth Bass		
Location	Hanover		

a. Concentration is expressed in ug contaminant per gram (wet weight) of fish

b. $$\text{Exposure level (mg/kg/day)} = \frac{\text{Concentration in fish (ug/g)} \times \frac{1\text{ mg}}{1000\text{ ug}} \times \text{Consumption Rate (g/day)} \times \text{Proportion of Exposure Duration (yrs/yrs)}}{\text{Body Weight (kg)}}$$

c. Estimated exposure is based on consumption rate of 7.8 g/day by a 70 kg adult and is an approximation of a "general consumer."

d. Estimated exposure is based on an approximated median consumption rate of 48 g/day by a 70 kg adult and is intended to represent a median consumption rate for an avid sports fishermen.

Both lead and methyl mercury are toxic to the central nervous system. It is therefore appropriate to assess their combined toxicity. Based on the lowest and highest measured concentrations, the resulting ranges of Hazard Indexes are as follows:

Consumption Rate	Low	High
7.8 g/day	2.4×10^{-2}	1.4×10^{-1}
48 g/day	1.5×10^{-1}	8.3×10^{-1}

These ranges of Hazard Indexes for people who consume either 7.8 g/day or 48 g/day are both below unity, indicating that there would be no anticipated adverse health effects as the result of combined exposure to lead and methyl mercury from consumption of the fish.

Of the four inorganic chemicals evaluated in this study, a FDA action level has been reported only for mercury (1 ppm; Table X). The range of measured mercury concentrations in these fish (<0.060 ug/g - 0.290 ug/g) is well below this 1 ppm (ug/g) FDA action level. These mercury concentrations are also below the more stringent 0.5 ppm action level reported by the State of Michigan (Humphrey et al, 1986).

3.4.2 - ORGANIC COMPOUNDS

DDT, DDE and DDD

Because of the similarities between DDT, DDE and DDD in chemical structure, target organ toxicity and cancer potencies, the DPHS believes it is appropriate to group these three chemicals as a class (referred to as "DDT/metabolites") for purposes of risk characterization.

The total concentration of DDT/metabolites is calculated by computing the sum of the concentrations of DDT, DDE and DDD for each composite sample (Table XIII). It is noted that DDE is the only chemical of these three that was consistently observed above the level of detection. Therefore, it is DDE's

Table XII - Ranges of Organic Chemical Concentrations in Connecticut River Fish and Corresponding Estimated Exposure Levels

Chemical	Range of Concentration[a]	Estimated Exposure Levels[b] (mg/kg/day) General Consumer[c]	Active Sports[d]
DDD[e,f]			
Lowest Conc.	0.010	1.11×10^{-7}	6.85×10^{-7}
Species	-[g]		
Location	All Locations		
Highest Conc.	0.02	2.23×10^{-6}	1.37×10^{-5}
Species	All Species		
Location	All Locations		
DDE[f]			
Lowest Conc.	0.001	1.11×10^{-7}	6.85×10^{-7}
Species	Yellow Perch		
Location	W. Lebanon		
Highest Conc.	0.260	2.90×10^{-5}	1.78×10^{-4}
Species	Walleye		
Location	Hanover		
DDT[f]			
Lowest Conc.	0.001	1.11×10^{-7}	6.85×10^{-7}
Species	-[g]		
Location	W. Lebanon		
Highest Conc.	0.02	2.23×10^{-6}	1.37×10^{-5}
Species	All Species		
Location	All Locations		
PCBs			
Lowest Conc.	0.031	3.45×10^{-6}	2.13×10^{-5}
Species	Chain Pickerel		
Location	Below Ashuelot		
Highest Conc.	1.640	1.83×10^{-4}	1.12×10^{-3}
Species	Walleye		
Location	Hanover		
PAHs			
Lowest Conc.	--	--	--
Species			
Location			
Highest Conc.	0.10	1.11×10^{-5}	6.86×10^{-5}
Species	All Species		
Location	All Locations		

a. Concentration is expressed in ug contaminant per gram (wet weight) of fish

b. $$\text{Exposure level (mg/kg/day)} = \frac{\text{Concentration in fish (ug/g)} \times \frac{1\text{ mg}}{1000\text{ ug}} \times \text{Consumption Rate (g/day)} \times \text{Proportion of Exposure Duration (yrs/yrs)}}{\text{Body Weight (kg)}}$$

c. Estimated exposure is based on consumption rate of 7.8 g/day by a 70 kg adult and is an approximation of a "general consumer."

d. Estimated exposure is based on an approximated median consumption rate of 48 g/day by a 70 kg adult and is intended to represent a median consumption rate for an avid sports fishermen.

e. Concentration was below the detection limit (BDL) for every sample taken.

f. Detection limit for this chemical was either 0.001 ppm or 0.02 ppm, depending on the particular sample.

g. This chemical was found BDL of 0.001 ppm in yellow perch, white perch, and smallmouth bass.

Table XIII

Range and Median Values for Cumulative DDD, DDE, and DDT Concentrations in Connecticut River Fish and Corresponding Estimated Exposure Levels

Detected DDT/ Metabolite Conc. (ug/g)[a]	Species/ Location	Consumption Rate (g/day)	Estimated Exposure Level[b,c] (mg/kg/day)
<.008 (Low Range)	S. Mouth Bass F./ W. Lebanon	7.8	$<8.91 \times 10^{-7}$
		48.0	$<5.49 \times 10^{-6}$
<.075 (median)	[d]	7.8	$<8.36 \times 10^{-6}$
		48.0	$<5.14 \times 10^{-5}$
<.300 (High Range)	Walleye F./ Hanover	7.8	$<3.34 \times 10^{-5}$
		48.0	$<2.06 \times 10^{-4}$

a. Concentration is expressed in ug contaminant per gram (wet weight) of fish

b. $$\text{Exposure level (mg/kg/day)} = \frac{\text{Concentration in fish (ug/g)} \times \frac{1\text{ mg}}{1000\text{ ug}} \times \text{Consumption Rate (g/day)} \times \text{Proportion of Exposure Duration (yrs/yrs)}}{\text{Body Weight (kg)}}$$

c. Esimated exposure is based on approximate consumption rate of either a general (7.8 g/day) or a high fish consumer (48 g/day).

d. Median value is actually an average of two mid-point values detected for walleye carcass sampled at Hanover and smallmouth bass carcass sampled at Claremont.

contribution to the overall concentration of DDT/metabolites which generally dictates the variability across samples.

As observed in Table XIII, the lowest DDT/metabolites concentration (<0.008 ug/g) was observed in smallmouth bass (fillets) sampled from the W. Lebanon location, while the highest concentration (<0.300 ug/g) was found in walleye (fillets) collected near Hanover. Other species observed to have comparatively high levels of DDT/metabolites include white perch (< 0.25 ug/g) and chain pickerel (<0.19 ug/g) carcasses collected below the Ashuelot River, and yellow perch carcasses (<0.180 ug/g) collected at the Claremont location. However, no consistent trend of concentrations was observed for any one particular species across all sampling locations. Since chain pickerel and walleye were each collected at only one of the five sampling locations, it is not known whether the high DDT/metabolite concentrations that were observed in these species are representative of other areas of the Connecticut River.

An intraspecies comparison of fillet versus carcass DDT/metabolite concentrations indicates that the latter were generally higher. This finding is in accordance with what is expected, since (as mentioned previously) hydrophobic DDT/metabolites are known to partition into fatty tissues. One of the few exceptions to this was the high level of DDT/metabolites detected in walleye fillets (<0.300 ug/g) compared to walleye carcasses (<0.070 ug/g). A comparison by sampling location indicates that the highest DDT/metabolite levels were generally observed in carcass samples collected below the Ashuelot River, as these samples contained anywhere from 0.140 ug/g to 0.250 ug/g.

If the result was reported as below the detection limit, it was assumed that the individual concentrations of DDD, DDE and DDT were equal to their respective limits of detection (0.02 ug/g for most samples). This assumption

was made since, theoretically, the concentration could be as high as the detection limit. In most cases this is likely to be an overestimate, since when reported as below detection, the actual concentrations of DDD, DDE or DDT could be as low as zero. For example, in fish collected from W. Lebanon, where a much lower detection limit equal to 0.001 ug/g was obtained for both DDD and DDT, only one of the six samples taken revealed that either of these chemicals were present at a level greater than 0.001 ug/g. If these W. Lebanon samples are representative of other locations, this would indicate that the actual concentrations of DDD and DDT could be well below 0.02 ug/g. Therefore, actual exposure levels to DDT/metabolites as a result of consumption of Connecticut River fish may approach a level which is equal to that of exposure solely to DDE.

Estimated ranges of exposure to DDT/metabolites are presented in Table XIII. The ranges of exposure to populations who consume either 7.8 g/day or 48 g/day are below the established oral RfD criterion for DDT (5×10^{-4} mg/kg/day), which indicates that noncarcinogenic health effects would not be expected.

Because the CPFs for DDD, DDE and DDT are very close quantitatively, it is considered appropriate by the DPHS to use a single CPF (0.34 mg/kg/day^{-1}) value to represent these three chemicals collectively. The equation used to estimate the upper-bound cancer risk level to an exposed population is as follows:

$$\text{Upper-bound Cancer Risk Level} = \text{Cancer Potency Factor } (\text{mg/kg/day})^{-1} \times \text{Exposure Level (mg/kg/day)}$$

The estimated upper-bound cancer risk is unitless, and is interpreted to represent the number of excess cancer cases resulting from exposure to a given

number of people. Based on the lowest, median and highest detected concentrations of DDT/metabolites, the estimated upper bound cancer risk levels to a population of people who consume 7.8 grams per day for 70 years are 3.0×10^{-7} (i.e., 3 excess cancers in 10 million people exposed), 2.8×10^{-6} and 1.1×10^{-5}, respectively (Table XIV). Based on the same low, median and high measured concentrations of DDT/metabolites in these fish, the estimated upper bound cancer risks to a population of people who consume a high amount of fish per day (48 g/day) are 1.9×10^{-6}, 1.8×10^{-5} and 7.0×10^{-5}, respectively (Table XIV).

Because our estimates of DDT/metabolite concentrations in fish are likely to be overestimates, the real cancer risks are likely to be much lower than reported here.

In characterizing the risk from exposure to DDT/metabolites as a result of Connecticut River fish consumption, it is recognized that the measured concentrations of DDE (which generally contribute a large proportion to the cumulative DDT/metabolite fish concentrations in this study) do not appear to be higher than concentrations which have been observed in fish sampled from other rivers in the northeast (Table IX).

The FDA has established an action level of 5 ppm (ug/g) for DDT/metabolites in fish (Table X). The range of DDT/metabolite concentrations observed in this study (0.008 ppm - 0.3000 ppm) falls well below this action level of 5 ppm, indicating that these fish would be acceptable in interstate commerce. However, since these action levels are not entirely health based, a certain level of risk may exist from consumption of fish that contain DDT/metabolite concentrations even below this FDA standard.

Table XIV
Estimated Population Cancer Risk Levels Based on Low, Median, and High Exposure Levels to Suspect Carcinogens

Chemical	Consumption Rate (g/day)	Estimated Exposure Level (mg/kg/day)[a,b]		Estimated Population Cancer Risk Level[c]
DDT/metab.[d]	7.8	Low	$<8.91 \times 10^{-7}$	$<3.0 \times 10^{-7}$
		Median	$<8.36 \times 10^{-6}$	$<2.8 \times 10^{-6}$
		High	$<3.34 \times 10^{-5}$	$<1.1 \times 10^{-5}$
	48.0	Low	$<5.49 \times 10^{-6}$	$<1.9 \times 10^{-6}$
		Median	$<5.14 \times 10^{-5}$	$<1.8 \times 10^{-5}$
		High	$<2.06 \times 10^{-4}$	$<7.0 \times 10^{-5}$
PCBs[e]	7.8	Low	3.45×10^{-6}	2.7×10^{-5}
		Median	3.12×10^{-5}	2.4×10^{-4}
		High	1.83×10^{-4}	1.4×10^{-3}
	48.0	Low	2.13×10^{-5}	1.6×10^{-4}
		Median	1.92×10^{-4}	1.5×10^{-3}
		High	1.12×10^{-3}	8.6×10^{-3}
PAHs[f]	7.8	Low	--	--
		Median	--	--
		High	$<1.11 \times 10^{-5}$	$<1.3 \times 10^{-4}$
	48.0	Low	--	--
		Median	--	--
		High	$<6.86 \times 10^{-5}$	$<7.9 \times 10^{-4}$

a. $$\text{Exposure level (mg/kg/day)} = \frac{\text{Concentration in fish (ug/g)} \times \frac{1\text{ mg}}{1000\text{ ug}} \times \text{Consumption Rate (g/day)} \times \text{Proportion of Exposure Duration (yrs/yrs)}}{\text{Body Weight (kg)}}$$

b. Various levels of exposure are based on low, median, and high concentrations detected in the fish sampled.

c. Estimated Population Cancer Risk = Exposure Dose (mg/kg/day) x CPF $(\text{mg/kg/day})^{-1}$.

d. The U.S. EPA reported CPF for DDT/metabolites = 0.34 $(\text{mg/kg/day})^{-1}$.

e. The U.S. EPA reported CPF for PCBs = 7.7 $(\text{mg/kg/day})^{-1}$.

f. The U.S. EPA reported CPF for B[a]P = 11.5 $(\text{mg/kg/day})^{-1}$. This CPF is for B[a]P, which is just one of the many chemical constituents which make up the total PAH concentration.

PCBs

The overall range of measured PCB concentrations in fish sampled across all species and locations was from 0.031 ug/g in chain pickerel fillets (below Ashuelot River; Table XII) to 1.64 ug/g in walleye fillets (Hanover; Table XII). A median PCB concentration equal to 0.28 ug/g was observed by computing an average of the two midpoint concentrations for smallmouth bass (carcasses) sampled at Claremont (0.26 ug/g) and W. Lebanon (0.30 ug/g). Samples other than walleye fillets which contain relatively high levels of PCBs include white perch carcasses (1.55 ug/g) and chain pickerel carcasses (0.95 ug/g) sampled from below the Ashuelot River. However, no consistent high trend of PCB levels was observed for any one particular species across all sampling locations. Since chain pickerel and walleye were collected at only one of the five sampling locations, it is not possible to determine from this data whether the high PCB concentrations are representative for the same species of fish which live in other areas of this River.

Due to their highly lipophilic properties, PCBs are typically expected to be present at higher concentrations in fatty areas (such as the skin) of fish. In fact, for most of the species that were sampled, the average PCB concentrations (across locations) were found to be higher in carcass compared with fillet portions. Walleye was the only species for which the average measured carcass PCB concentration (0.210 ug/g) was lower than in fillets (1.640 ug/g).

Since there is no reported oral RfD for PCBs, a comparison of estimated exposures with this criterion cannot be made to estimate the non-carcinogenic health risks.

Based on the lowest, median and highest measured concentrations of PCBs (across all species and locations sampled) the estimated upper bound cancer risk levels to a population of people who consume 7.8 g/day are 2.7×10^{-5}, 2.4×10^{-4} and 1.4×10^{-3}, respectively (Table XIV). Based on these same low, median and high PCB fish concentrations, the estimated cancer risks to a population which consumes 48 g/day are 1.6×10^{-4}, 1.5×10^{-3} and 8.6×10^{-3}, respectively (Table XIV). Therefore, general and high fish-consuming populations may be exposed to an increased cancer risk from consumption of these Connecticut River fish, as the result of PCB exposure.

However, as discussed previously, these measured PCB concentrations in Connecticut River fish do not appear to be any higher than those levels which have been reported in other rivers (Table VIII). For example, while the highest measured concentration of PCBs in this study is 1.64 ug/g in walleye, there have been reported PCB concentrations for this same species as high as 2.78 ug/g in other states's freshwaters (Table VIII).

The FDA has established a tolerance level for PCBs in fish equal to 2 ppm (Table X). The range of PCB concentrations observed in this study (0.031 ug/g - 1.64 ug/g) is below this tolerance level, indicating that these Connecticut River fish would be suitable to use for interstate commerce. However, since tolerance levels are not entirely health based, a certain level of risk may exist from consumption of fish that contain PCBs even below this FDA standard.

PAHs

All of the fish sampled in this study were observed to contain total PAH concentrations below the level of detection (0.10 ug/g). Since the concentration of PAHs could theoretically be as high as the detection limit, as a worst case scenario, exposure levels were calculated based on consumption

of fish that contain 0.10 ug/g total PAHs. As observed in Table XII, the estimated PAH exposures to a population of people who consume 7.8 g fish/day and 48 g fish/day are $<1.11 \times 10^{-5}$ mg/g/day and $<6.86 \times 10^{-5}$ mg/kg/day, respectively.

PAH metabolites, some of which are carcinogenic, were not included in these analyses for this study. While metabolite levels in flesh may be high enough to warrant concern, this cannot be determined from the data. It is noted that in one composite sample of yellow perch collected more recently, there was a low level of PAHs in the carcass, making it theoretically possible that PAH metabolites could be present in the flesh.

Because no oral RfD that has been reported by the U.S. EPA for PAHs, a comparison cannot be made to determine whether there would be any predicted non-carcinogenic risk.

Since B[a]P is only one of the many constituents present in PAHs, by setting an acceptable risk based on this constituent it is assumed that B[a]P's cancer potency is representative of all PAH constituents as a whole. It is noted that a total of 18 different PAH chemical constituents were included in these laboratory analyses in order to represent a total measured PAH concentration. However, only 6 of these 18 constituents have been classified by the U.S. EPA as group B carcinogens (probable human carcinogens). Most of the PAH constituents have been designated by U.S. EPA as group D carcinogens (not classifiable as to carcinogenicity), or were not assigned to any group classification due to the paucity of relevant scientific data. Since the non-carcinogenic constituents could make a significant contribution to the total PAH concentration, the actual CPF for PAHs as a whole may be much lower than the potency factor for B[a]P. This assumption will therefore overestimate the true risk.

Assuming the cancer potency for B[a]P (11.6 mg/kg/day^{-1}), the resulting estimated upper bound population cancer risk levels are reported as follows:

a) population of people who consume 7.8 grams/day, cancer risk = 1.3 x 10^{-4};

b) population of people who consume 48 grams/day, cancer risk = 7.9 x 10^{-4}

(Table XIV). However, the actual concentrations of PAHs in Connecticut River fish are present at some unquantifiable level below 0.10 ug/g. Therefore, the true cancer risk posed to a population of people who consume these fish is likely to be much lower than the above calculations.

3.5 SUMMARY

The available data indicate that consumption of Connecticut River fish may result in exposure to at least some of the chemicals surveyed in this study. In an attempt to characterize the potential health risks from these exposures, both carcinogenic (cancer-causing) and non-carcinogenic health effects were evaluated.

Some general assumptions were made in the process of assessing both the carcinogenic and non-carcinogenic health risks from consuming Connecticut River fish. For example, it was assumed that: 1) the measured chemical concentrations in fish surveyed are representative of those present in fish that are actually caught and consumed by local fishermen; 2) that such daily consumption occurs over a person's lifetime and consists entirely of fish taken from the study reach of Connecticut River; 3) that avid sports fishermen consume approximately 48 grams of fish per day and the general population consumes 7.8 grams of fish per day.

The above assumptions introduce a large amount of uncertainty into the risk assessment. In order to be protective of public health, the assumptions made

are designed to be conservative, and are more likely to err on the side of protecting public health by overestimating risk rather than underestimating risk.

In order to model a worst-case scenario, additional conservative assumptions were used to estimate cancer risk levels. For example, the calculated cancer risks are based on an upper-bound 95% confidence interval estimate, which is an innately conservative estimate. Also, when concentrations of chemicals such as DDT/metabolites and PAHs were reported below their detection limits, these were assumed to be equal to their respective detection limits. In summary, all of the above mentioned factors will lead to an overestimated cancer risk.

Estimated exposure levels from consumption by either the general fish consumer or by the avid sports fisherman were not found to pose any significant non-carcinogenic health risks.

Population cancer risk levels were estimated for the three different suspected carcinogenic organic chemicals analyzed, including DDT/metabolites, PAHs and PCBs. Based on the range of concentrations of DDT/metabolites, the estimated range of population cancer risks to people consuming 7.8 and 48 grams of fish per day are 3×10^{-7} - 1.1×10^{-5}, and 1.9×10^{-6} - 7.0×10^{-5}, respectively. However, the actual ranges of risks from exposure to DDT/metabolites may be much lower, since it was assumed that concentrations reported as below detection were equal to it.

For PAHs, the upper-bound cancer risk estimate was calculated based on the cancer inducing potency of B[a]P, which is just one of the many chemical constituents that make up this class of chemicals. Based on the detection

limit concentration for PAHs, the estimated population cancer risks to individuals consuming 7.8 and 48 grams of fish per day are 1.3×10^{-4} and 7.9×10^{-4}, respectively. However, the actual risks may be much lower if the other PAH constituents are less toxic than B[a]P or vice versa.

Based on the range of concentrations of PCBs, the estimated range of population cancer risks to people who consume 7.8 and 48 grams of fish per day are $2.7 \times 10^{-5} - 1.4 \times 10^{-3}$, and $1.6 \times 10^{-4} - 8.6 \times 10^{-3}$, respectively. These ranges of cancer risks are likely to be reasonable estimates and both do exceed the maximum acceptable risk of 10^{-5}. Although consumption of these fish may pose a significant cancer risk from PCB exposure, the concentrations of PCBs in most species of fish sampled were not any higher than corresponding species sampled from other rivers in northeastern states. The highest PCB concentration (1.64 ug/g) was detected in walleye fillets sampled at Hanover. This concentration approaches the FDA tolerance level of 2 ppm, but does not exceed it. Therefore, a person could potentially be exposed to higher PCB levels in fish that are purchased at the supermarket or restaurant. Also, since walleye fillets were only collected at one of the five sampling locations, it is not possible to conclude whether this result was representative of walleye that live in other parts of the River.

As observed in Table XIV, of the various suspect carcinogens for which cancer risk levels were determined, PCBs were estimated to contribute the greatest risk. Based on average PCB concentrations in the fillet (skin off) of each species, the number of consumed meals per year that correspond to increased risk levels of 10^{-6}, 10^{-5} and 10^{-4} were determined (Table XV). As illustrated in Table XV, to avoid an increased risk of no more than 1×10^{-6}, one needs to restrict consumption to less than one meal per year for all species sampled, except chain pickerel. Consumption of no more than one meal per year of chain pickerel is indicated to avoid exceeding this same risk.

The most remarkable fillet PCB concentration was observed in the walleye collected at Hanover, which contained a level of 1.64 ppm. This PCB concentration, which approaches the FDA tolerance level, is based on a single composite collected at Hanover, and therefore may not be representative of levels present in walleye living in other areas of the Connecticut River. Therefore, it is recommended that further walleye sampling be conducted at a representative number of locations to verify whether this single composite sample is representative of walleye in other areas of the River.

TABLE XVI

Increased Risk of Cancer Associated with Consumption Frequencies for Each Species

Frequency of Meal Consumption[a]	Increased Risk of Cancer		
	Chain Pickerel	Smallmouth Bass	Walleye
1 meal/year	1 in 1,000,000	1 in 100,000	7 in 100,000
2 meals/year	3 in 1,000,000	1 in 100,000	1 in 10,000
3 meals/year	4 in 1,000,000	3 in 100,000	2 in 10,000
6 meals/year	8 in 1,000,000	6 in 100,000	4 in 10,000
12 meals/year	2 in 100,000	1 in 10,000	9 in 10,000
2 meals/month	3 in 100,000	2 in 10,000	2 in 1,000
1 meal/week	7 in 100,000	5 in 10,000	4 in 1,000
1 meal/day	5 in 10,000	4 in 1,000	3 in 100
Frequency of Meal Consumption	White Perch	White Sucker	Yellow Perch
1 meal/year	2 in 100,000	3 in 1,000,000	9 in 1,000,000
2 meals/year	4 in 100,000	7 in 1,000,000	2 in 100,000
3 meals/year	5 in 100,000	1 in 100,000	3 in 100,000
6 meals/year	1 in 10,000	2 in 100,000	6 in 100,000
12 meals/year	2 in 10,000	4 in 100,000	1 in 10,000
2 meals/month	4 in 10,000	8 in 100,000	2 in 10,000
1 meal/week	9 in 10,000	2 in 10,000	5 in 10,000
1 meal/day	6 in 1,000	1 in 1,000	3 in 1,000

a. It is assumed that one meal of fish weighs one third of a pound, or 150 grams.

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APPENDIX A

QUALITY ASSURANCE/QUALITY CONTROL
METALS IN TISSUE

CADMIUM

1. Blank Data

Blank Number	Results (ug/g)
MB 246	0.003

2. Accuracy

Sample Number	Original Concentration (ug/g)	Spike Level (ug/g)	Total Concentration Found (ug/g)	% Recovery
8860-1	0.16	0.50	0.6	88
8860-2	0.12	0.49	0.56	90

3. Precision

Sample Number	Rep 1 (ug/g)	Rep 2 (ug/g)	Average (ug/g)	% Rel. Range
8860-1	0.16	0.15	0.16	6.3
8860-2	0.096	0.14	0.12	37

QUALITY ASSURANCE/QUALITY CONTROL
METALS IN TISSUE

CHROMIUM

1. Blank Data

Blank Number	Results (ug/g)
MB 246	0.1

2. Accuracy

Sample Number	Original Concentration (ug/g)	Spike Level (ug/g)	Total Concentration Found (ug/g)	% Recovery
8860-1	0.69	1.00	1.5	81
8860-2	0.96	0.98	1.96	102

3. Precision

Sample Number	Rep 1 (ug/g)	Rep 2 (ug/g)	Average (ug/g)	% Rel. Range
8860-1	0.59	0.78	0.69	28
8860-2	0.88	1.03	0.96	16

QUALITY ASSURANCE/QUALITY CONTROL
METALS IN TISSUE

LEAD

1. Blank Data

Blank Number	Results (ug/g)
MB 246	0.01

2. Accuracy

Sample Number	Original Concentration (ug/g)	Spike Level (ug/g)	Total Concentration Found (ug/g)	% Recovery
8860-1	<0.5	5.00	4.9	98
8860-2	<0.5	4.9	4.2	86

3. Precision

Sample Number	Rep 1 (ug/g)	Rep 2 (ug/g)	Average (ug/g)	% Rel. Range
8860-1	<0.5	<0.5	<0.5	---
8860-2	<0.5	<0.5	<0.5	---

QUALITY ASSURANCE/QUALITY CONTROL
METALS IN TISSUE

MERCURY

1. Blank Data

Blank Number	Results (ug/g)
HgB 5	0.0000

2. Accuracy

Sample Number	Original Concentration (ug/g)	Spike Level (ug/g)	Total Concentration Found (ug/g)	% Recovery
8860-1	0.10	0.70	0.43	47
8860-2	0.20	0.96	0.59	41

3. Precision

Sample Number	Rep 1 (ug/g)	Rep 2 (ug/g)	Average (ug/g)	% Rel. Range
8860-1	0.087	0.12	0.10	33
8860-2	0.21	0.19	0.20	10

QUALITY ASSURANCE/QUALITY CONTROL

PESTICIDES/PCB'S

1. Blank Data

Blank Number	Results (ug/g)
B 486	<.01 (PCB's) <.001 (Pesticides)

2. Accuracy

Sample Number	Original Concentration (ug/g)	Spike Level (ug/g)	Total Concentration Found (ug/g)	% Recovery
S-465 (filet)	.167	.20	.256	45
S-466 (filet)	.167	.19	.292	65

3. Precision

Sample Number	Rep 1 (ug/g)	Rep 2 (ug/g)	Average (ug/g)	% Rel. Range
Filets spiked at .2 ug/g	.256	.292	.274	13

QUALITY ASSURANCE/QUALITY CONTROL

POLYNUCLEAR AROMATIC HYDROCARBONS

1. Blank Data

Blank Number	Results (ug/g)
B 486	<.1

2. Accuracy

Sample Number	Original Concentration (ug/g)	Spike Level (ug/g)	Total Concentration Found (ug/g)	% Recovery
S-465 (filet)	<.1	12.8	10.9	85
S-466 (filet)	<.1	12.8	11.6	89

NOTE: The spike solution contained 16 individual Polynuclear Aromatic Hydrocarbons at equal concentrations of .8 ug/g each.

3. Precision

Sample Number	Rep 1 (ug/g)	Rep 2 (ug/g)	Average (ug/g)	% Rel. Range
Filets spiked at 12.8 ug/g	10.9	11.6	11.3	6.2

APPENDIX B

WEIGHTED CONCENTRATION CALCULATIONS

$$\left[\begin{array}{l}\text{Sample} \\ \text{weight}\end{array} \times \begin{array}{l}\text{Tissue} \\ \text{concentration} \\ \text{of contaminant}\end{array}\right]_{\text{offal}} + \left[\begin{array}{l}\text{Sample} \\ \text{weight}\end{array} \times \begin{array}{l}\text{Tissue} \\ \text{concentration} \\ \text{of contaminant}\end{array}\right]_{\text{fillet}} = \begin{array}{l}\text{Mass} \\ \text{of} \\ \text{cont.}\end{array}$$

$$\text{Mass of contaminant} \;/\; \text{Sample weight}_{\text{offal}} + \text{Sample weight}_{\text{fillet}} = \begin{array}{l}\text{Weighted} \\ \text{concentration} \\ \text{(Whole body)}\end{array}$$